

The Australian and New Zealand Journal of Surgery

OCTOBER, 1938.

TABLE OF CONTENTS.

[The Whole of the Literary Matter in THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY is Copyright.]

ORIGINAL ARTICLES—

	PAGE
ABRAHAM COLLES AND HIS FRACTURE	<i>K. F. Russell</i> 115
THE ANATOMY OF THE BRONCHIAL TREE AND ITS CLINICAL APPLICATION <i>J. Hardie Neil, W. Gilmour, F. J. Gwynne, Wallace Main and W. A. Fairclough</i>	118
INTRACRANIAL ANEURYSMS, WITH SPECIAL REFERENCE TO SURGICAL TREATMENT <i>E. Graeme Robertson</i>	132
A REVIEW OF THE ACUTE POST-OPERATIVE CIRCULATORY DISTURBANCES <i>Sir Hugh Devine</i>	145
THE SURGICAL TREATMENT OF PROSTATIC OBSTRUCTION: A NINE-YEAR REVIEW FROM THE NEWCASTLE HOSPITAL	<i>Thomas Hamilton</i> 156
TRANSURETHRAL PROSTATIC RESECTION: THE INDICATIONS FOR ITS PERFORMANCE AND ITS RESULTS	<i>M. S. S. Earlam</i> 161
A NOTE ON THE VARIATIONS OF THE FORAMEN OVALE	<i>Sydney Sunderland</i> 170
EARLY INDICATIONS FOR SURGERY IN HEAD INJURIES	<i>I. Douglas Miller</i> 176
PNEUMOCOCCAL PERITONITIS	<i>A. Eisdell Moore</i> 182

SURGICAL TECHNIQUE—

THE USE OF THE DIATHERMY LOOP IN THE TREATMENT OF PILONIDAL SINUSES <i>A. Eisdell Moore</i>	186
--	-----

CASE REPORTS—

MULTIPLE DUODENAL, INTESTINAL AND OTHER ABNORMALITIES ..	<i>Hugh R. G. Poate</i> 188
ACTINOMYCOSIS OF THE VERTEBRAL COLUMN	<i>Richard Flynn and A. D. Gillies</i> 193
LARGE THORACTIC GANGLIO-NEUROMA	<i>J. Maxwell Clarke</i> 199

EDITORIAL—

THE PATHOLOGICALLY-MINDED SURGEON	203
---	-----

SURGERY IN OTHER COUNTRIES—

KERATOPLASTY	205
SPONDYLOLISTHESIS	207
CAROTID-CAVERNOUS ANEURYSMS	210
THE BLOOD PHOSPHATASE IN PATIENTS SUFFERING FROM CARCINOMA	211
THE BLOOD PHOSPHATASE IN PREGNANCY	212
SECONDARY HYDATID CYSTS OF THE PLEURA	212
OPERATIVE TREATMENT OF HALLUX VALGUS	213
INTRACRANIAL LESIONS IN RETROBULBAR NEURITIS	214

REVIEWS—	PAGE
INJURIES AND DISEASES OF THE HIP	215
APPENDICITIS	216
SURGICAL PATHOLOGY OF DISEASES OF THE NECK	216
A SURGEON'S POCKET BOOK	217
 PROCEEDINGS OF THE ROYAL AUSTRALASIAN COLLEGE OF SURGEONS—	
QUEENSLAND ANNUAL MEETING	218
NEW ZEALAND ANNUAL MEETING	218
VICTORIAN ANNUAL MEETING	219
TASMANIAN STATE ANNUAL MEETING	220
SOUTH AUSTRALIAN ANNUAL MEETING	220
VICTORIAN EVENING MEETINGS	220
REGISTRATION OF THE COLLEGE DIPLOMA OF FELLOWSHIP	221
GORDON CRAIG LIBRARY	221
GORDON CRAIG SCHOLARSHIPS	221
 NOTICES	222
 EDITORIAL NOTICES	222

THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY

Registered at the General Post Office, Melbourne, for transmission by post as a periodical.

VOL. VIII.

OCTOBER, 1938.

No. 2.

ABRAHAM COLLES AND HIS FRACTURE.

By K. F. RUSSELL,
Melbourne.

ABRAHAM COLLES, the second son of Mr. William Colles, was born in 1773 at Milmount on the banks of the River Nore near Kilkenny. He received his early education in the endowed school of Kilkenny, and in 1790 he entered the University of Dublin to pursue his medical course. During this time he was apprenticed to Dr. Woodroffe, then a resident surgeon in Steeven's Hospital. While still a student he wrote a paper "On the Condition of Political Satire", which earned the recommendation of Edmund Burke, but was never published. In 1795 he obtained his diploma from the Royal College of Surgeons in Ireland, and almost immediately went to Edinburgh, where he obtained his M.D. degree.

Then followed a tour to London, where he visited all the principal hospitals. Having a great interest in anatomy, Colles became acquainted with Sir Astley Cooper (Mr. Cooper as he was then), and he made the dissections from which the plates for Astley Cooper's folio on hernia were engraved. During this friendship with Cooper, Colles conceived the idea of writing a book on surgical anatomy. This work, however, was never finished, and the first part only was published in 1811.

In 1797 he returned to Dublin and became a candidate for the position of resident surgeon in Steeven's Hospital, which post he obtained in 1799. While in this position he inaugurated the system of giving regular clinical



A. Colles

lectures to his students. He then rapidly became a leading member of the surgical profession of Dublin, as a study of his income shows. In 1798 he received in fees £8 10s. 7½d., in 1799-1800 he received £178 4s. 4d., in the following year his receipts were £421 7s. 5d., and in 1826, at the height of his career, his fees amounted to £6,128.

In 1802 he sought the Chair of Anatomy and Surgery in Trinity College, but was defeated by Dr. Hartigan. In 1804 the chair again fell vacant, and Colles was appointed to it in conjunction with Mr. Dease. This chair he held for no less than thirty-two years. His health commenced to decline in 1835, and in the following year he was forced to resign his chair. His health did not improve, and he died on November 16, 1843, in his seventy-first year.

Throughout his entire career as a clinical teacher he lost no opportunity of frankly admitting his blunders, using them, in fact, to instruct his students. He twice occupied the presidential chair of the Royal College of Surgeons in Ireland, in 1802 and 1830, and in his latter years was offered a baronetcy on more than one occasion, which offer, however, he firmly declined.

In his writings he took the greatest interest in syphilis and its treatment, in which he advocated small doses of mercury. He showed that the disease was infective during the secondary stage as well as the primary, and wrote much on its signs and symptoms both in children and adults. He wrote a paper "On the Operation of Tying the Subclavian Artery", which was published in 1815. In this paper he advocated the ligation of the artery on the medial side of the *scalenus anticus* muscle. Garrison, in his "History of Medicine", states that Colles is said to have been the first in Europe to tie the innominate artery successfully, but he is unable to verify this assertion. Colles also wrote a paper on the "Fatal Consequences Resulting from Slight Wounds Received in Dissection", in which he quotes cases which occurred amongst his own students.

In 1814 there appeared in Volume x of *The Edinburgh Medical and Surgical Journal* a short paper of four pages in which Colles presented to the world his classical description of the fracture which now bears his name. This paper, "On the Fracture of the Carpal Extremity of the Radius", is at the present time probably the only one of his writings by which Colles is known. In it he presents in a very lucid manner the underlying pathology of the fracture and suggestions for its treatment.

It would not be out of place if one were to quote at some length portions of this paper:

The fracture takes place at about an inch and a half above the carpal extremity of the radius and exhibits the following appearances. The posterior surface of the limb presents a considerable deformity; for a depression is seen in the forearm, about an inch and a half above the end of the bone, while a considerable swelling occupies the wrist and metacarpus; indeed the carpus and base of the metacarpus appear to be thrown backwards. . . .

There is a fullness on the anterior surface of the limb which extends upwards to about one-third of the length of the forearm and terminates below at the upper end of the annular ligament of the wrist. The extremity of the ulna is seen projecting towards the palm and inner edge of the limb.

If the surgeon proceeds to investigate the nature of the injury . . . he will discover on the posterior surface, by touch, that the swelling on the wrist and metacarpus is not caused entirely by an effusion among the softer parts; he will perceive that the ends of the metacarpus and second row of carpal bones form no small part of it . . . when he moves his fingers along the anterior surface of the radius he finds it more full and prominent than is natural. When he attempts to move the

broken pieces of bone in opposite directions, although the patient is by this examination subjected to considerable pain, yet neither crepitus nor a yielding of the bone at the seat of the fracture, nor any other positive evidence of the existence of such an injury, is thereby obtained. If the surgeon lock his hand in that of the patient and make extension, even with a moderate force, he restores the limb to its natural form, but the distortion of the limb instantly returns on the extension being removed. . . .

The unfavourable result of some of the first cases which came under my care forced me to investigate the nature of the injury. . . . Let the surgeon apply the fingers of one hand to the seat of the suspected fracture, and, locking the other hand in that of the patient, make a moderate extension until he observes the limb restored to its natural form. As soon as this is effected let him move the patient's hand backward and forward, and he will, at every such attempt, be sensible of a yielding of the fractured ends of the bone. . . . The hard swelling which appears on the back of the hand is caused by the carpal surface of the radius being directed slightly backwards instead of looking directly downwards. The carpus and metacarpus, retaining their connection with this bone, must follow it in its derangements, and cause the convexity above alluded to. . . .

It is obvious that in the treatment of this fracture our attention should be principally directed to guard against the carpal end of the radius being drawn backwards.

For this purpose, while assistants hold the limb in a middle state between pronation and supination, let a thick and firm compress be applied transversely on the anterior surface of the limb at the seat of fracture, taking care that it shall not press on the ulna; let this be bound on firmly with a roller, and then let a tin splint, formed in the shape of the arm, be applied to both its anterior and posterior surfaces. . .

It is scarcely necessary to observe that the two principal splints should be much more narrow at the wrist than those in general use, and should also extend to the roots of the fingers, spreading out so as to give a firm support to the hand.

The cases treated on such a plan have all recovered without the smallest defect or deformity of the limb.

The portrait of Colles appearing at the commencement of this article is taken from the New Sydenham Society publication mentioned below.

BIBLIOGRAPHY.

Robert McDonnell: "Selections from the Works of Abraham Colles", London, New Sydenham Society, 1881.

Fielding H. Garrison: "An Introduction to the History of Medicine", 4th Edition, W. B. Saunders and Company, 1929.



THE ANATOMY OF THE BRONCHIAL TREE AND ITS CLINICAL APPLICATION.¹

By J. HARDIE NEIL, W. GILMOUR, F. J. GWYNNE, WALLACE MAIN
AND W. A. FAIRCLOUGH.

[From the Throat, Pathological, Radiological and Ophthalmic Departments
of the Auckland Hospital.]

THE advances made during recent years in the diagnosis by radiology, and in the treatment by surgery, of lung diseases has stimulated the study of the anatomy of the bronchial tree.

After the trachea has divided into the two primary bronchi, the secondary bronchi to the upper, middle and lower lobes are, of course, recognized by their well-known constancy. When the tertiary bronchi are under consideration, their apparent inconstancy has caused anatomists to fall back on the terms "dorsal", "lateral" and "ventral", which are frequent in text-books on vertebrate comparative anatomy. In some of the vertebrates, for example the pig, the tertiary bronchi of the lower lobe can be seen coming off from the dorsal, lateral, ventral and mesial aspects in regularly diminishing size, from before backward or cephalo-caudad. The stem apparently branches monopodially to the final main terminal bifurcation. In the human being some of the tertiary bronchi, to which we shall refer later, individualize themselves to form a characteristic bronchial tree, whilst others found in more primitive types of lung disappear. The terms "dorsal", "lateral" and "ventral" were used in human anatomy to describe the location of the lower lobe bronchi, whose orifices appeared in corresponding surfaces of the bronchial stem. These terms were meaningless so far as ultimate distribution was concerned and carried no salient memorizing data. Their apparent inconstancy prevented precision in description, and their further distribution was seemingly lost in a maze. As an example, Dwight Davis,⁽¹⁾ after describing the secondary bronchi according to their possession of two or three branches, finished a paper dealing meticulously with over eighty specimens by stating:

Since the arrangement of the bronchi is so varied, it is difficult to reach a definite conclusion, except that each lung is different. No other organ, so far as I am aware, has such a varied arrangement of its fundamental structures.

He used radiographs of lungs into the bronchi of which opaque media had been forced.

Kramer and Glass,⁽²⁾ in 1932, published what we consider to be a most important paper. They have shown that the tertiary bronchi, or those that come off the main bronchi, are distributed with a constancy of 85%, and that

¹ This paper formed the basis of a demonstration at the annual meeting of the Royal Australasian College of Surgeons at Auckland on January 14, 1937.

each tertiary bronchus is distributed to a special segment of the lung, forming what they term a broncho-pulmonary segment, whose limits can be delineated on the periphery, and whose relations to the chest wall can be defined. In all the lungs that we have examined (apart from one in which the right upper lobe came off the trachea as in some herbivora) our findings for what we term in the text as consonants are almost 100% constancy. The subapicals

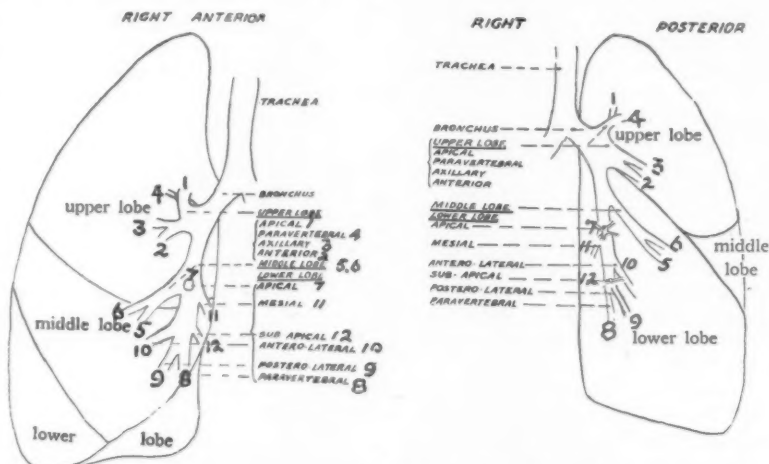
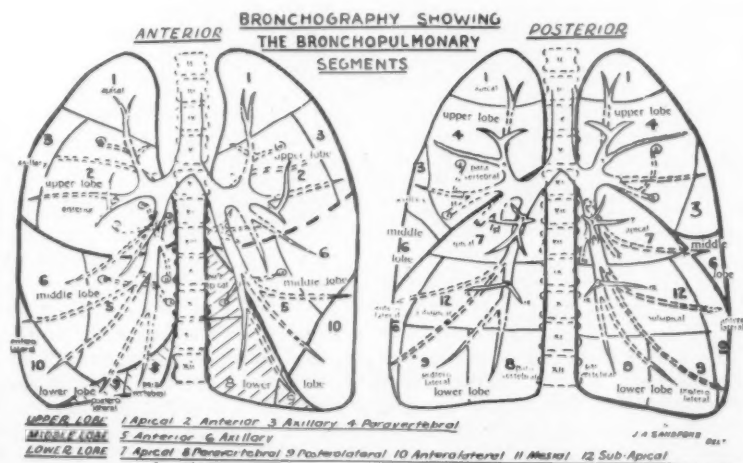


FIGURE I. This figure shows the origins of the tertiary bronchi and gives corresponding numbers followed in subsequent diagrams



ADAPTED FROM KRAMER & GLASS.

FROM THE THROAT PATHOLOGICAL & X RAY DEPT'S

AUCKLAND HOSPITAL

FIGURE II. The broncho-pulmonary segments.

are the variants. Kramer and Glass, in their investigation, made use of liquid dyes in fresh inflated lungs obtained at autopsy. Nelson,⁽³⁾ in 1934, published an article in which his findings were in consonance with those of Kramer and Glass.

Let us now consider Figure I, which is a simple diagram of the bronchial tree, and Figure II, which shows the distribution of the broncho-pulmonary segments. These illustrations show our amendments to the findings of Kramer and Glass. These workers found that the constant bronchi of the main stem of the bronchial tree were those of the upper lobe, of the middle lobe and of the lower lobe; those of the lower lobe, with which we are dealing specially at this time, included the apical, the mesial (or cardiac or infra-cardiac, which is found only on the right side), the antero-lateral and the terminal para-vertebral and postero-lateral. It will be noted that the terminology of Kramer and Glass conveys a topographical description which allows a ready visualization of the architecture of the lungs. Their anatomical findings can be readily correlated with the rudimentary bronchial buds in the lungs of a nine-millimetre embryo.

With the exception of the mesial, all the broncho-pulmonary segments have surfaces in contact with the chest wall, and are therefore accessible to surgery. The mesial or cardiac segment is so constant and accessible to bronchoscopy that surgical requirements for drainage can be met by a suction tube.

Kramer and Glass make a most important statement, that pathological change in the lung due to aspiration infection is primarily confined to one broncho-pulmonary segment. In lung abscesses, the pathological process makes its way towards the periphery, setting up an adhesive pleurisy which leads to adhesion of the segment involved to the chest wall. With a knowledge of broncho-pulmonary anatomy, the surgeon can make an incision through adhesions safely, open a lung abscess and avoid the disaster of opening the pleural cavity or, what is worse, of incising normal lung tissue.

TECHNIQUE.

In our investigations into bronchial anatomy, we made use of fusible metal in autopsy specimens of human and other mammalia, the dissection and inflation of autopsy specimens, the examination of the lung *in vivo* by bronchoscopy, by means of which we injected lipiodol into the individual segments separately. We used Wood's fusible metal, which consists of one part of tin, one part of cadmium, two of lead and four of bismuth. Thrice this quantity in ounces is a useful amount for a large adult human lung. This compound will melt in boiling water, but as it quickly cools and will produce only stumpy casts if used at 100° C. (212° F.), we found this method useless. In making casts of human lungs, the heating of the metal after the components are fused should be continued for at least another minute over an ordinary Bunsen burner. This will give a temperature of approximately 148° C. (300° F.). In small mammals the extra heating may cause the pleura to be perforated. In these cases, when the metal is fused and the dross is cleared, a temperature of about 121° C. (250° F.) is satisfactory. The thorax of the animal should be unopened, and the funnel used in the trachea should have a metal tube down within the spout, to allow the gases to escape from the trachea and to avoid spluttering of the metal, which is introduced

in a steady stream. After a few minutes the lungs are removed, and as much lung tissue as possible is removed from the lung surface, care being taken not to interfere with the terminations of the cast. Surface incisions are made into the lung, the bronchial branches being avoided. The specimen



FIGURE III. The right bronchial tree opened from the front, showing the upper and middle orifices and the orifices of the broncho-pulmonary segments of the lower lobe.

is then immersed in a 25% solution of caustic soda for forty-eight hours. With toothed forceps the tissues can then be removed by pulling them off in the axes of the bronchi. Such specimens will afford an intimate knowledge of the anatomical points, and by holding them in appropriate positions, whilst viewing specimens and radiographs, one may clarify an apparently hopeless problem.

Dissection and inflation can be carried out in fresh autopsy specimens. For making a permanent specimen for demonstration of inflation of separate

segments the Mooltan⁽⁴⁾ method is advisable. The apparatus is simple and can be arranged in any laboratory. After the specimen is hardened by the frequent filling of the tree by 5% formalin for two or three days, it should be taken out of the vacuum, drained and the tree filled with methylated spirit, which should be gently forced in so as to fill the tree in its medium-sized branches. After two hours it should be drained, or suction applied, and allowed to lie submerged in the formalin solution for a few days. Suction or drainage will give the specimen gaping orifices. A Eustachian catheter



FIGURE IV. The left bronchial tree shown as in Figure III.

attached to the Higginson syringe will permit inflation even of the branches of the tertiary bronchi, and accurate demarcation of the segments. Failing the Mooltan method, the specimen can be put into 5% formalin solution for twenty-four hours, after some of the solution has been forced into the bronchial tree with a syringe. It is then drained trachea downwards, or treated by suction. Wrapping specimens in the following preserving fluid will keep them soft and pliable.

Two hundred grammes of arsenate of soda are boiled in two litres of water and added to seven litres of tap water in which a kilogram of acetate of soda has been dissolved. Three litres of glycerine are then added.

For dissection of the bronchi, as shown in Figures III and IV, blunt-pointed curved scissors will give access to the plane of tissue round the bronchi. By teasing and finger dissection, all the branches mentioned can be readily displayed. They should be exposed for about an inch. If the main bronchi are incised prior to this, the specimen may be spoilt, as orientation may be lost. We have found that by the use of a Higginson syringe and a

suitable nozzle, inflation through the bronchus of each pulmonary segment will cause it to project on the surface of the lung, with clear demarcation. The use of lipiodol has certainly advanced the study of bronchography. Unfortunately, the method generally used is to instil amounts varying from five to fifteen cubic centimetres down the trachea, or into a bronchus, and to take radiographs. The result is a superimposition of bronchial shadows which may reveal gross pathological change, but the separate broncho-pulmonary segments can rarely be definitely determined, unless one side only is filled and a lateral view taken. We have adopted the method of single filling of the separate bronchial branches, after they have been cleared of discharge by suction, restricting the amount to one cubic centimetre for

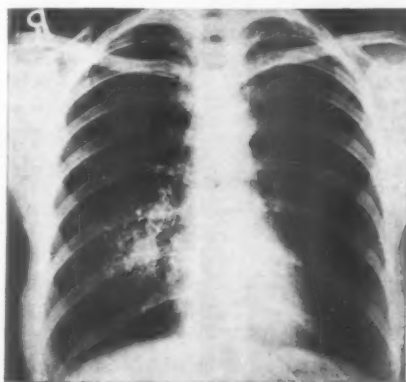


FIGURE V. A single filling of the apical segment of the right lower lobe.

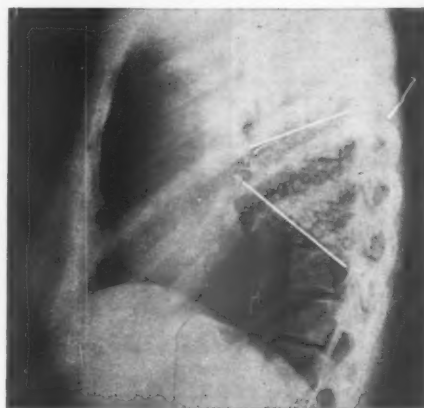


FIGURE VI. A lateral view of Figure V.

each. The patient is then sent immediately to the radiological department, where posterior to anterior and lateral views are taken. The illustrations will show how necessary views in two planes are for proper orientation (see Figures V and VI).

RESULTS OF INVESTIGATION.

We may summarize the results of our investigations as follows. The upper lobe bronchus divides constantly into four tertiary bronchi supplying the apical, paravertebral, anterior and axillary segments. The paravertebral broncho-pulmonary segment, the bronchus of which is the first to come off from the dorsal aspect of the vestibule, is much larger than has hitherto been recorded. It is also, by the way, a site of predilection for invasion by tuberculosis. The middle lobe is served by a bronchus which constantly divides into an anterior and an axillary branch.

We have been concentrating our investigations on the variations of the lower lobe bronchi. The apical bronchus of the lower lobe is consistently present. Indeed, it has been frequently shown that it supplies a separate or partially separate lobe. The mesial or infracardiac bronchus is constant again, and the same statement applies to the antero-lateral bronchus, which

may, however, have two openings: the anterior cephalad and the lateral caudad. The bronchial tree ends by dividing into two terminal bronchi, the inner or mesial paravertebral and the outer or lateral postero-lateral. Our principal new finding is that in varying positions between the apical branch above and the terminal branching below are found one or two bronchi which have apparently escaped notice by all anatomists, surgeons and pathologists to whose writings we have had access.

It is necessary to refer to comparative anatomy to obtain an explanation of their occurrence. In the cat we see a bronchial tree which has anatomical constants that are unequivocally homologous with the bronchi of the broncho-

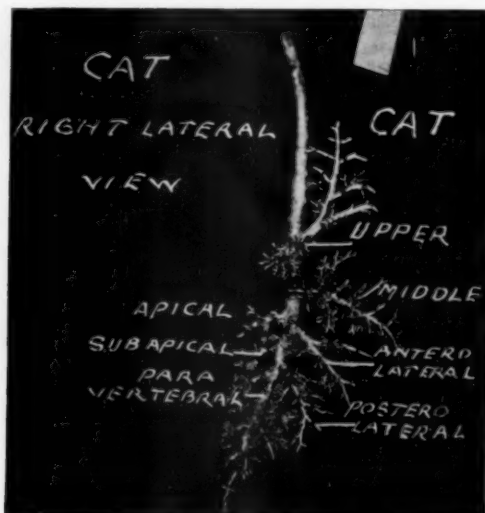


FIGURE VII. A Wood's metal cast of the right lung of a cat.

pulmonary segments detailed above in the human subject (see Figure VII). From and including the apical bronchus of the lower lobe, there is a series of sprouts or branches in descending magnitude down to the tips of the terminal bronchial branches. They are mainly on the dorsal and lateral surfaces, except in the terminal bronchi, where they are also seen on the ventral surface, with one or two on the mesial. Between the apical branch and the final division of the stem into the paravertebral and postero-lateral branches there is constantly found on the dorsal aspect a well-marked sprout on both the right and the left stems. In our earliest communication⁽⁵⁾ we termed this the posterior intermediate. Unfortunately, this term is used in descriptions of one of the air sacs, apart from the lungs proper, that occur in birds. The name "subapical" has therefore been adopted as a better term. Homologues of this may be seen in the mammalia that we have had the opportunity of examining, *i.e.*, the monkey, cat, sheep, pig, deer, kangaroo, wombat, koala, dog, rabbit and opossum.

In a series of metal casts of some mammalian lungs that we have prepared, the dorsal branches of the lower main stem bronchi are seen in regular sequence. Beneath or caudad to the first or apical bronchus, the homologue of the human subapical bronchus is constantly seen above the final bifurcation into the paravertebral and postero-lateral branches. In a specimen from the opossum two subapical branches were found on the dorsal aspect and two on the mesial. The distribution of the subapical to the area



FIGURE VIII. Single filling of the right subapical segment.

between the apical bronchus above and the paravertebral below is constant. In the human left lung we have found it situated on the mesial aspect, or the dorsal, or at times lower down on the dorsal aspect just at the orifice of the paravertebral bronchus. Twice clinically we have found it on the lateral and twice on the ventral aspect. This is in accord with our findings in some of the mammalia as previously mentioned, where homologues of the subapical branch may be found on all four aspects of the bronchial tree. In one or two cases it has been absent. The significance of this in the human is not clear at present. It may be that it is a recessive variation. However, on the right side, where there may be two separate openings, we have found it constantly just below the apical bronchus on the dorsal surface, mesially below the mesial, or on the dorsal or lateral aspect, just above the final

bifurcation of the stem. Any specimen apparently at variance with this statement should have the various orifices inflated. We have proved by means of metal casts, inflation, lipiodol instillation and radiography its distribution to the territory between the apical bronchus above and the paravertebral below, and naturally have found it to be the subject of pathological change. In the latest published contributions to the anatomy and pathology of the lower lobe it has been incorporated in the paravertebral segment. We suggest that with knowledge of the varying sites of the subapical bronchi there are



FIGURE IX. . Lateral view of Figure VIII.

no obstacles in the way of the description of the lung in terms of bronchopulmonary segments with the degree of error permitted in anatomical description (see Figures VIII and IX).

With more intimate anatomical knowledge, clinical work will be more precise, and further investigation can be made into the predilection for certain sites to be affected in such diseases as tuberculosis, bronchiectasis and lung abscess. Three of the authors of this paper have published an article⁽⁶⁾ that will give data on topography which will be of interest to radiologists, because in it single fillings of segments are shown (see Figures X and XI).

The infracardiac or mesial bronchus, as we term it, is well marked as supplying a separate lobe in our specimens of the monkey, cat, pig, duiker, sheep, dog and kangaroo. In the human being it is incorporated in the right lung. The late Sir Colin MacKenzie, of the Australian Institute of Anatomy at Canberra, kindly sent us drawings of the findings in the wombat, as seen

in Figures XII and XIII, in which it is well developed, and of those in the koala or native bear in which it is apparently absent. On examination of the lungs of a specimen of each, sent to us by Sir Colin, the wombat had the usual mammalian cardiac lobe, but in the koala no external cardiac lobe was found. On opening the bronchial tree, a bronchial orifice was found in the lower lobe in the usual position of the mesial or cardiac bronchus in the other mammals of our series. Inflation outlined an area similar to that

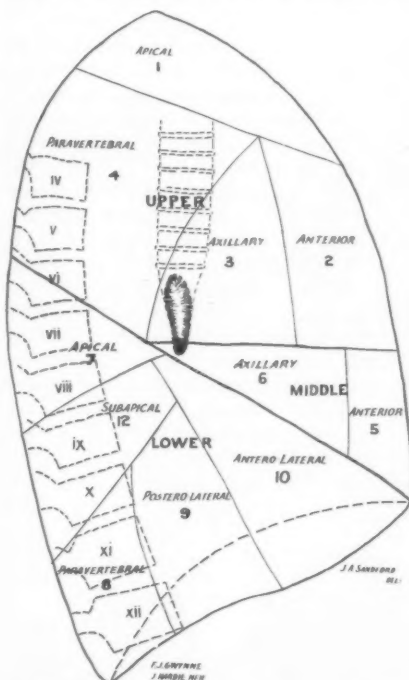


FIGURE X. Lateral view of right lung, showing surface projection of broncho-pulmonary segments, composed from single fillings. If this illustration is enlarged by pantograph onto a tracing paper, so that the transverse diameter through the level of the eighth dorsal vertebra measures about seven and a half inches, it can be placed over a standard film and the various broncho-pulmonary segments delineated. A used film, cleared by dipping in boiling water, makes an efficient tracing sheet.

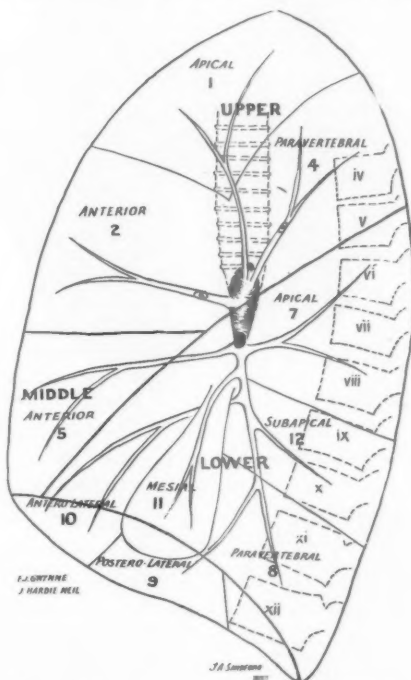


FIGURE XI. Mediastinal view of Figure X.

found in the human subject. The koala, therefore, is the only animal in our series that, like the human being, has its mesial or cardiac lobe incorporated in the right lung (see Figures XIV and XV).

CLINICAL APPLICATION OF BRONCHOGRAPHY.

We shall now discuss the clinical application of bronchography (see Figure XVI). As the other bronchi mentioned are constants, the orifices of the subapical bronchi are the variants which may give rise to non-recognition or confusion. The identification of a bronchus is of great importance, as the

orifice of a diseased segment generally shows indications of disease. Septic foci, such as abscesses and infected tuberculous cavities, are frequently met with. Their cure or relief is greatly hampered by obstruction of the bronchi leading to them. Disease products and mucus frequently so hamper the action of the all-important cilia, which lift the mucus and *débris* upwards,

that the cavity becomes practically closed, and destructive anaerobic bacteria may increase the broken-down area surrounding the cavity.

There is no known drug which, in therapeutic doses, can alter the activity of the cilia, and mucus cannot be either dissolved or absorbed in the body. Hence comes the importance of opening up or clearing the affected bronchi. In suction we have an important means of attaining this end. In dealing with the infection of cavities, the removal of *débris* must be our main objective. Antiseptics, though useful, must play a minor part compared with that played by the potent connective tissue cells or macrophages which line the air sacs and detach themselves from the walls to engulf foreign matter or *débris*.

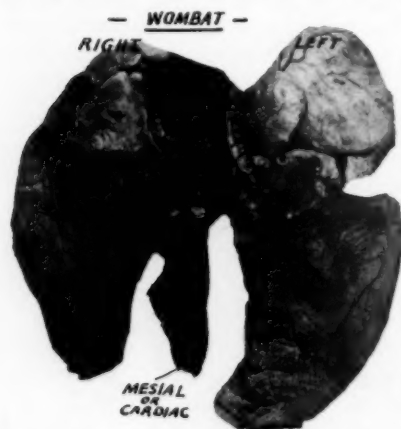


FIGURE XII. Lung of wombat showing cardiac lobe.

Antiseptic injections may be used. Solutions of an aqueous isotonic type are much more effective than oily solutions, and are apparently harmless. Experiments on dogs have shown that isotonic solutions, with the equivalent of the salt content of the blood, can, by drop flow, be instilled into the bronchi up to the amount of six litres, without apparently causing harmful changes in the general body conditions, or subsequent serious microscopic lesions in the lungs. Coloured aqueous solutions have been found to have passed through the finer bronchi into the air sacs and onto the rich lymphatic system. The fluid has not returned by the trachea. Use is now made of this fact to deal with abscess and bronchiectatic cavities. Once the

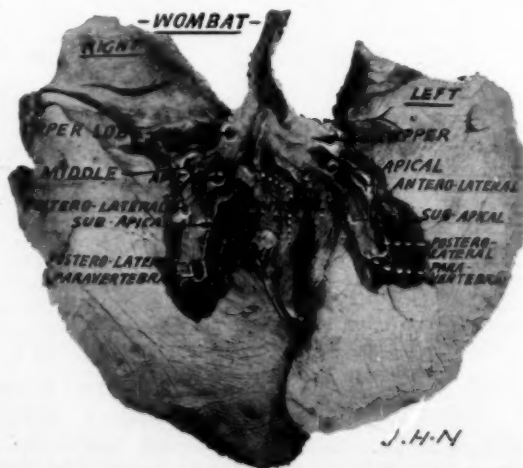


FIGURE XIII. Lungs of wombat showing bronchial tree opened from the front.

bronchi leading to them, and if possible the cavities themselves, are emptied by suction, suitable antiseptics in aqueous solution can be introduced in amounts varying from 30 to 300 cubic centimetres. A Murphy drip is used at the rate of one drop in two seconds. Some of the more potent antiseptics that have been introduced in the last few years are now available. We have made use of "Merthiolate" and "Metaphen" in the strength of one in 10,000 in Martin Bledsoe irrigant, as mentioned by Kolmer.⁽⁷⁾ "Merthiolate" and "Metaphen" are organic mercurial compounds which will in laboratory tests, in a dilution of one in 60,000 in the presence of serum, destroy the streptococcus in five minutes. They are thus most potent destroyers of the *Streptococcus hæmolyticus*, which is the most common pyogenic organism in lung disease. In therapeutic doses they are apparently innocuous to the kidneys. Martin Bledsoe irrigant consists of:

Sodium chloride	263 grammes
Potassium chloride	10 grammes
Calcium chloride (desiccated)	21 grammes
Distilled water	1,000 cubic centimetres

Seven and a half cubic centimetres (2 drachms) of the above solution are added to 170 cubic centimetres (2 ounces) of distilled or recently boiled

water at a temperature of 37.6° C. (100° F.). One should commence by using 56.7 cubic centimetres (2 ounces) and work up to 170 cubic centimetres (6 ounces), at weekly intervals. Bronchoclysis is the name given to this procedure by Mandelbaum.⁽⁸⁾ He introduced a catheter into the lower part of the bronchial stem. We have shown by cinema that we can, by means of the bronchoscope, introduce the catheter into the bronchus of the exact segment involved. The radiographs taken subsequently

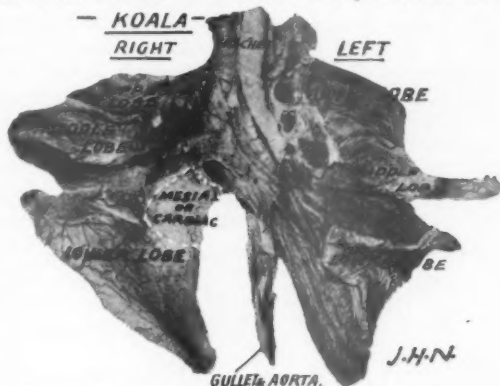


FIGURE XIV. Lungs of koala showing the cardiac lobe fused with the right lung as in the human being.

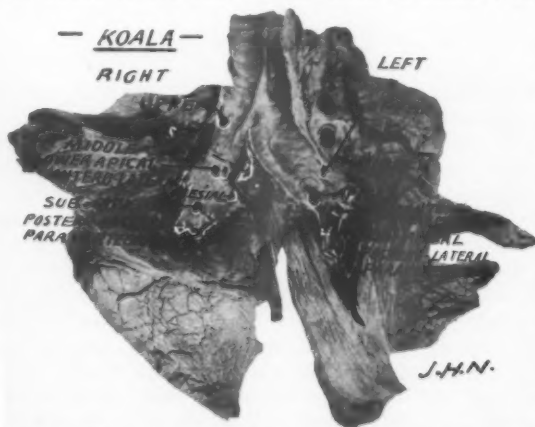


FIGURE XV. Lungs of koala with bronchial tree opened from the front. On the right side the middle lobe bronchus has been cut across as it lies in front or ventral.

show a cloudiness of the lung tissue that disappears within a week, when the procedure may be repeated. Lower respiratory diseases bulk largely in mortality statistics, and contribute largely to disabilities entailing serious economic inefficiency.

Speaking generally, and disregarding the congenital and rarer forms mentioned in text-books on thoracic surgery and medicine, the bronchiectasis found in the everyday work in clinics commences during the infectious respiratory diseases of childhood. It is first noted by physical and radiological examination in the paravertebral and postero-lateral terminal branches of the bronchial tree. Thence it creeps or spills into the neighbouring bronchi. The initial infection very frequently involves simultaneously the upper and lower

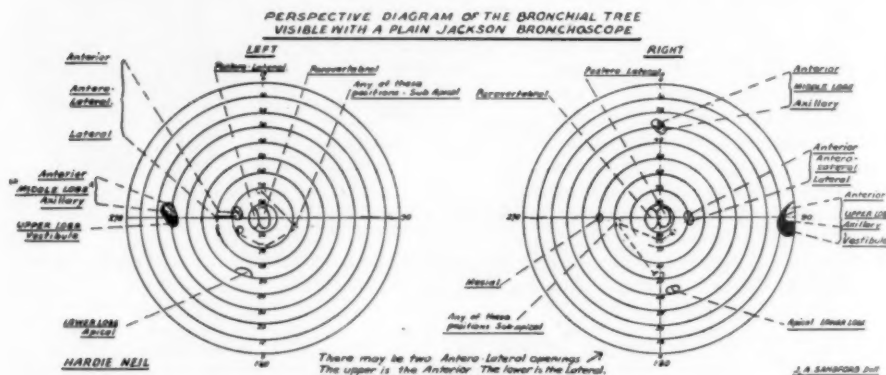


FIGURE XVI. A perimetric perspective view showing the relative positions of the bronchial orifices.

respiratory tracts. If the sinus infection clears up, the lung has such recuperative power that it will prevent the establishment of bronchiectasis, unless severe and special local damage has occurred, such as may take place from bronchopneumonia or from constant reinfection from the sinuses. A child with recurring bronchitis should have a radiological and clinical examination of its sinuses and lungs. Evidence of sinusitis and bronchitis should call for immediate treatment. Opening of the antra, with removal of a sufficient amount of the inferior turbinate bones to ensure permanent ventilation, is a minor operation, and bronchial aspiration is also a minor procedure. If the medical profession can become familiar with the anatomy of the bronchi and realize that bronchoscopic inspection and drainage are innocuous and effective, a great advance will have been made in the prophylaxis of this disease, which is more common than tuberculosis, blights and shortens so many lives, and frequently lies hidden under the diagnosis of chronic bronchitis. We are grateful to the pioneers for their work in this special branch of medicine, but trust that others will add to their efforts and help to minimize the incidence of respiratory diseases, in which chronicity is an ever-increasing menace to relief.

SUMMARY.

Reference is made to the difficulty that exists in describing the bronchial branches in terms used in comparative anatomy.

The apparent inconstancy of the tertiary bronchi is deemed to be a cause of the failure of text-books to standardize the anatomy of the bronchial tree.

The work of Kramer and Glass is elaborated and a separate area, the subapical, of the lower lobe of the lung is described. It has a bronchial supply hitherto unrecorded. The bronchi of this area are located, and their homologues shown in mammalia other than the human being. The recognition of this bronchus and its variants allows the anatomy of the bronchial tree to be described with the surety of ordinary anatomical description.

New methods are described of making metal casts of the bronchial tree, of showing the distribution of the bronchi by inflation, and of filling single bronchi by means of lipiodol.

A method is described of instilling into the lung antiseptics in isotonic solution in large amounts that are absorbed by the lymphatics.

A plea is made for the recognition of bronchiectasis in early life, and suggestions are offered for its preventive treatment.

ACKNOWLEDGEMENTS.

We wish to express our indebtedness to Mr. J. A. Sandford, who made the line drawings for us. He thinks that he is repaid by having had a staple removed that had been in his lung for thirty-six years. The staffs of the radiological and pathological departments of the Auckland Hospital have also given valuable assistance.

REFERENCES.

- (1) J. Dwight Davis: "Anatomic Variations of the Normal Tracheo-Bronchial Tree", *Archives of Otolaryngology*, Volume ix, April, 1929, page 404.
- (2) R. Kramer and A. Glass: "Bronchoscopic Localization of Lung Abscess", *Annals of Otology, Rhinology and Laryngology*, Volume xli, December, 1932, page 1211.
- (3) H. P. Nelson: "Postural Drainage of the Lungs", *The British Medical Journal*, Volume ii, August 11, 1934, page 251.
- (4) Sylvan E. Mooltan: "A Simple Apparatus for Fixation of the Lungs in an Inflated State", *Archives of Pathology*, Volume xx, July, 1935, page 77.
- (5) J. Hardie Nell, Walter Gilmour and Wallace Main: "Proceedings of Second Pan-Pacific Surgical Congress", August, 1936.
- (6) J. Hardie Nell, W. Gilmour and F. J. Gwynne: "The Broncho-Pulmonary Segments: Radiological, Pathological and Bronchoscopic Considerations, with Special Reference to the Subapical Broncho-Pulmonary Segment", *The Medical Journal of Australia*, July 31, 1937, page 165.
- (7) J. A. Kolmer: "Bronchial Disinfection and Immunization", *Archives of Internal Medicine*, Volume li, March, 1933, page 692.
- (8) M. J. Mandelbaum: "Bronchoclysis", *Archives of Physical Therapy, X-Ray and Radium*, Volume xlii, November, 1932, page 673.

INTRACRANIAL ANEURYSMS, WITH SPECIAL REFERENCE TO SURGICAL TREATMENT.¹

By E. GRAEME ROBERTSON,

*Honorary Physician to Out-Patients, Royal Melbourne Hospital;
Honorary Physician to the Austin Hospital.*

[From the Walter and Eliza Hall Institute of Research in
Pathology and Medicine, Melbourne.]

SIR WILLIAM OSLER⁽¹⁾ states that sunstroke is one of the oldest of recognizable diseases, and cites the illness of the son of the Shunammite woman, described in the second book of Kings. However, in those few vivid verses the rupture of an intracranial aneurysm is as readily recognizable as in any long clinical history:

And when the child was grown, it fell on a day, that he went out to his father to the reapers.

And he said unto his father, My head, my head. And he said to a lad, Carry him to his mother.

And when he had taken him and brought him to his mother, he sat on her knees till noon, and then died.²

The period of apparent death and the restoration of life to the child by the prophet Elisha surely measured the duration of the loss of consciousness which so frequently occurs in this condition. No other description of the severe headache of instantaneous onset, indicative of rupture of intracranial aneurysms, need be given. It is the purpose of this paper to deal with the question of the surgical treatment of congenital aneurysms.

The presence of an aneurysm is usually suspected only after it has ruptured, but the larger ones may produce symptoms and signs in virtue of the fact that they are space-filling lesions, and for this reason are sometimes diagnosed as neoplasms and operated upon, probably more frequently than is recognized. In 1923, Cushing was able to find records of only three cases of aneurysm for which operation had been carried out; but this proves little beyond the fact that it is only our successes that we are willing to blazon abroad. Sometimes calcification within the wall of the aneurysm or in the laminated clot throws a ring-like shadow in skiagrams (Albl's ring), and so the nature of the tumour can be diagnosed. Collier reports a case in which Sir Percy Sargent removed one of two calcified masses in the suprapituitary region, this proving to be one lobe of a clotted and calcified aneurysm. The memory of a skiagram (Figure I) seen during student days was responsible for the correct posthumous diagnosis of a

¹ Delivered at the tenth annual general meeting of the Royal Australasian College of Surgeons held at Auckland in January, 1937.

² II Kings, Chapter iv, verses 18, 19, 20.

lesion which produced paresis of the left-sided third cranial nerve, left-sided headache and right-sided hemiplegia of twelve years' duration.

Examination revealed ptosis of the left upper eyelid, loss of upward movement of the left eye and rigidity of the right arm and leg, without reflex or sensory changes. In spite of the reported absence of reflex changes, review of a cinema film of the patient's gait taken by Dr. Douglas Thomas suggests that the motor state was due in part at least to involvement of the pyramidal tract. It is probable that there was both pyramidal and extrapyramidal involvement. The patient was operated upon



FIGURE I. Albl's ring: a circular shadow seen in skiagram due to the deposition of calcium salts in the periphery of an aneurysm in which lamination with clot has occurred.

by Sir Alan Newton, and the tumour, thought to be a meningioma, removed. The patient died after operation, and the *post mortem* record merely remarks upon disintegration of the lower and middle parts of the left temporal lobe, "as though it had been deprived of its blood supply".

There is no doubt that the tumour was an aneurysm situated on the left side of the circle of Willis.

Although it is common for such calcified aneurysms to produce signs and symptoms of local cortical irritation, or more usually of impairment of function, aneurysms causing such symptoms are by no means usually calcified.

M.P., a male, aged twenty-five years, was admitted to the Royal Melbourne Hospital on February 3, 1936, under the care of Dr. R. P. McMeekin. For ten years the patient had suffered from intermittent frontal headaches, which increased in intensity some six months before admission, and became associated with soreness behind the eyes. In October, 1935, he commenced to suffer from prolonged bouts of severe pain behind the left eye, whence the headache seemed to travel to the anterior part of the left temple, where it was of throbbing character. Early in November, 1935, he commenced to suffer from "smelling turns", an acrid smell commencing in the upper part of the left nostril and increasing with each breath, merging into a feeling of dizziness. At the same time something would flash through his mind, about a person he had known long ago to whom he said something which he never could recall. He afterwards wondered why he thought of that person, although he could



FIGURE II. Aneurysm growing from a point of bifurcation of the middle cerebral artery in the Sylvian fissure, and impinging upon the medial part of the temporal lobe (uncinate region). The inferior surface of the frontal lobe, medial to the aneurysm, has been cut away.

never recapture details nor even recollect who the person was, although he was sure he was "someone very familiar a long time ago". The attacks usually occurred as he was in the act of falling asleep each night, and afterwards he felt so bright that he would lie awake for hours. During the last week in November the attacks were very frequent, and in one attack he slumped in his chair and was unable to prevent his relatives carrying him from the room.

The only abnormality revealed by repeated examination was a constant diminution in the right-sided superficial abdominal reflexes. The pressure of the cerebro-spinal fluid was 190 millimetres of water. It contained twenty lymphocytes, two large mononuclear cells and twelve erythrocytes per cubic millimetre. There was no increase of globulin and no reaction to the Wassermann test.

Of the localization of the lesion there could be little doubt; the description of the uncinate epilepsy could not be clearer. The patient unfortunately died after surgical exploration, in the course of which a hard pulsating mass was palpated in the region of the left uncus.

At autopsy, subjacent to the tip of the uncus, there was a round aneurysm, with a fibrous wall and filled with blood clot, whose neck communicated with the middle cerebral artery in the depths of the Sylvian fissure between the frontal and temporal lobes (see Figure II).

Aneurysms are probably a commoner cause of focal epilepsy than is generally realized.

The record of the following patient suggests that the hæmorrhage from an aneurysm may be mistaken for that due to trauma. Indeed, it is possible that trauma may initiate hæmorrhage from an aneurysm.

A boy, ten and a half years of age, was known by his doctor, Dr. A. P. Derham, to have a congenital lesion of his heart, probably a patent interventricular septum, which had caused rapid fatigue and slight dyspnoea on exertion. On May 15, 1935, the patient fell down some steps onto the right side of his head, face and body. He cried, but made little fuss about it. He seemed quite well the next day, but on May 17, after going to a party, he complained of severe pain in the right side of the head. He slept well and the next day he had no headache, but had no inclination for food. On May 19, at 9.30 a.m., he was riding a tricycle, and his brother told his mother that he had "gone mad in the head". His mother found him clammy, wet and complaining of severe headache. He vomited many times and intermittently screamed with pain in the head. After lunch he asked his mother to hold the right side of his head and fell back in her arms, and afterwards remained unconscious, with intermittent twitching of his left arm and both legs.

He was first seen by Dr. Derham at 5.30 p.m., and was found to be in a state of cerebral irritation. His temperature in the groin was 36.1° C. (97° F.), and his systolic blood pressure was 180 millimetres of mercury. The right pupil was larger than the left, both reacting sluggishly to light. He had a bilateral Kernig's sign and his plantar responses were equivocal. When seen at 6.15 p.m. he was cyanosed and had ceased breathing. All limbs were flaccid, with absent deep reflexes, and both plantar responses were extensor in type.

Artificial respiration was commenced, and it was considered that a carefully performed lumbar puncture might reduce the intracranial pressure sufficiently to allow respiration to recommence, although it was probable that the respiratory failure was due to the formation of a pressure cone. Blood-stained fluid rose to a height of 50 millimetres in the manometer, so the needle was withdrawn. Lumbar puncture probably carried with it a risk of death which should not have been taken. Cardiac stimuli were given, and he was transferred to hospital, artificial respiration being continued from its inception till the death of the patient at 4.30 a.m. At 7 p.m. Dr. A. E. Coates made a trephine hole over the right parietal region and disclosed a thin layer of blood in the subarachnoid space. A needle was then passed into the right lateral ventricle and blood under very high pressure was evacuated. A decompression opening was made, but respiration did not recommence. No fluid could be obtained from the left ventricle. Decompression of the medulla by removal of the arch of the atlas and bone surrounding the *foramen magnum* had no effect in restoring respiration.

At autopsy a ruptured aneurysmal sac, about 0.83 centimetre (one-third of an inch) in diameter, was found growing from a point of bifurcation in the middle cerebral artery in the depths of the Sylvian fissure between the frontal and temporal lobes. The escaping blood had ploughed up cerebral tissue, and had reached the right lateral ventricle, which was distended with blood clot. The foramen of Munro on the left side was blocked by blood clot and the left lateral ventricle was small and contained only a little blood-stained fluid.

The question of the treatment of aneurysms is of great importance. There has been considerable discussion as to the rôle of repeated lumbar puncture in the treatment of subarachnoid hæmorrhage, the opponents of this procedure believing that it increases the hæmorrhage. Lowering the intracranial pressure, which has been so acutely raised, causes a lowering of blood pressure which probably more than counteracts the effect of the lessening of cerebro-spinal fluid pressure in increasing the blood loss. The cardinal fact is that these patients do not die of exsanguination; they die of increased intracranial pressure, perhaps combined with the effects of shock and meningeal irritation.

Lumbar puncture is usually performed to confirm the diagnosis, the pressure of the cerebro-spinal fluid being lowered to within normal limits (120

millimetres of cerebro-spinal fluid, as measured by a manometer). The procedure is repeated should the pulse become markedly slow or the patient complain of unbearable headache or lapse into unconsciousness, and will frequently serve to increase the pulse rate, relieve headache and restlessness and restore consciousness. Morphine is valuable in this, as in other forms of hæmorrhage. In 1923 Harvey Cushing apologized for writing a note upon intracranial aneurysms, a subject having, as he said, such remote surgical bearing, yet in 1888 Gowers⁽²⁾ mentions the operation of ligation of the common carotid artery to promote clotting in aneurysm of the internal carotid artery within the skull. Beadles⁽³⁾ states that Horsley, in 1902, performed a similar operation and the patient was alive and well five years later. In 1928 Natrass⁽⁴⁾ reported a successful operation of ligation of the internal carotid artery by Professor Grey Turner for an aneurysm upon that artery. In 1927 Moniz⁽⁵⁾ showed that visualization of the intracranial circulation by radiography following the injection of a radio-opaque substance ("Thorotrast") was a practical procedure, apparently free from risk, in living persons. In 1932 Dott⁽⁶⁾ described several cases in which this procedure was used for localization of aneurysm, and in which surgical treatment was undertaken. The cases were divided into two groups, one in which the aneurysm was upon the internal carotid artery (proximal to the circle of Willis) and the other in which it was upon, or distal to, the circle of Willis.

The latter group may be dealt with first. Dott describes one successful case in which a piece of muscle was applied to the site of rupture in an attempt to induce clotting which would later be followed by repair by fibrous tissue. No further reports of such an operation have been published, and the question as to whether anything may be gained by surgical interference frequently arises. Of thirty-eight patients diagnosed as suffering from intracranial aneurysm at the Royal Melbourne Hospital since 1929, twenty have perished, two having survived previous ruptures, and eighteen have survived. No doubt many of these will die as a result of a second rupture, but some will die from other causes. The mortality is high enough in all conscience, but can it be lowered by surgical treatment?

The symptoms of rupture of an intracranial aneurysm are those of effusion of blood into the subarachnoid space, and there are other, albeit rarer, causes of subarachnoid hæmorrhage. One patient, suffering from her fourth subarachnoid hæmorrhage, was shown after death to have had a primarily intraventricular hæmorrhage from a capillary angioma. Dr. R. J. Wright-Smith tells me that in 1,351 *post mortem* examinations performed for the coroner of the City of Melbourne, he found diffuse subarachnoid hæmorrhage, without a visible aneurysm and without a history of trauma, in twelve. In fourteen cases an aneurysm was found to be the cause. It is probable that some of the twelve cases were of aneurysm, but if it was not found at autopsy it would scarcely be found at operation. Subarachnoid hæmorrhage may result from a tumour whose presence has been previously unsuspected, and from aneurysms other than those of congenital origin, such as arterio-sclerotic and mycotic aneurysms. Lateralizing signs are frequently absent and, unless they afford evidence of impairment of cranial nerves, are liable to be misleading. Even after injection of "Thorotrast" into both internal carotid arteries an aneurysm may remain unlocalized, as in one proven at autopsy to be upon the anterior communicating artery. If the aneurysm

be upon the basilar system, where it rarely is, injection of the vertebral artery would also be necessary.

The youth of the patient, the degree of head retraction, much more marked than that found in adults, and the location of the aneurysm make the next case worth describing in some detail.

G.K., a boy, aged four years, was admitted to the Children's Hospital, Melbourne, on November 19, 1936, under the care of Dr. A. P. Derham. Three days before admission he was picked up by his ankles and swung around by a maid. He did not bump his head, but when let go he walked unsteadily and lay down under a table, complaining of severe headache. Soon afterwards he vomited, and a quarter of an hour later he became unconscious and remained so for twenty-four hours, vomiting at intervals. After recovering consciousness he complained of frontal headache, and his mother noticed that his neck was stiff and that he was feverish.

Examination revealed very marked retraction of the head, great cervical rigidity and Kernig's sign. All deep reflexes were present and equal, the abdominal reflexes were present, and the plantar responses were extensor. The cerebro-spinal fluid was under increased pressure and deeply blood-stained.

Lumbar puncture was repeated at intervals, and although the cerebro-spinal fluid was brightly blood-stained and under a pressure of more than 300 millimetres on November 25, it was merely yellow, with an initial pressure of 70 millimetres, on November 28, 1936. His clinical state improved coincidentally, but he died of lobar pneumonia on December 5, 1936. Autopsy revealed a small clotted aneurysm 0.5 centimetre (one-fifth of an inch) in diameter, growing from the point of bifurcation of the left posterior inferior cerebellar artery upon the postero-lateral aspect of the medulla.

No reference has yet been made to the difficulty of finding the aneurysm at operation when the field is obscured by blood. If the aneurysms be multiple, obliteration of one sac will not prevent subsequent hæmorrhage from other sources. If the aneurysm be situated upon the middle cerebral artery deep in the Sylvian fissure or on the anterior cerebral artery between the frontal lobes, it may rupture intracerebrally, when much difficulty would be encountered in finding the aneurysm. If, as in another case, the patient is apparently dying and has a block in the cerebro-spinal fluid circulation (shown by Queckenstedt's test), apparently due to the formation of a pressure cone, then ventricular tapping or perhaps decompression would be justified. The trauma of "Thorotrast" injection and operation may well weigh down the pan of an already precariously balanced scale. If the aneurysm were located it might be possible to ligate its neck, but that would be possible only in very fortunate circumstances. Ligation of the internal carotid artery for aneurysm upon that artery has been performed, probably on many occasions, by Dott and other surgeons, including Lendon,⁽⁷⁾ of Adelaide, who has demonstrated the position of the aneurysm by injection of "Thorotrast" and successfully ligated the internal carotid artery in four patients. No untoward results following this operation have been recorded, except in one patient in whom, as stated by Albright,⁽⁸⁾ Dandy ligated the internal carotid artery for an aneurysm upon the middle cerebral artery. Death was due to the pressure of the aneurysm upon the middle cerebral artery, the blood flow in which was already depleted by ligation of the internal carotid. In this operation surgery has a very proper place in the treatment of intracranial aneurysm.

The following case is of extreme interest in view of the fact that an unhappy result was obtained, and it is described in detail because of the many lessons that can be learnt from it.

L.R., a woman, twenty-five years of age, was admitted to the Royal Melbourne Hospital on June 19, 1936. On admission she stated that while defæcating twelve hours before she had felt something burst in her head and had lost consciousness, recovering a short time later with violent headache over the head and at the back of the neck. At a later date she was able to amplify the history, stating that intermittently during the previous twelve months she had experienced a "sore" pain above her eyes when she awakened in the morning. This pain would gradually disappear in the early afternoon. During the last three months of this period, during moments of irritation, she felt as though there were a "bunch of nerves which wanted to burst" in the centre of her forehead.

On the night of June 18, 1936, her baby was restless and prevented her from sleeping, and during the waking hours she felt the sensation as of impending bursting inside her head for a much longer period than ever before. At 9 a.m., while straining at stool, being unusually constipated, she felt something burst inside her head, and experienced a shocking pain in the centre of her forehead as though she had been struck there. This was followed by a sensation of something trickling down inside her forehead and over the back of her eyes. In trying to get out of the lavatory she collapsed on the floor and next remembers seeing a clock pointing to 9.30 a.m. She felt a very intense, throbbing headache on top of her head, as though she were being hit there with a hammer. She was very alarmed to find that she could not move her legs, which felt cold, but after her husband had applied a hot water bottle they became normal. Dry retching commenced at midday and continued during the rest of the afternoon, during which time pain was also developing at the back of the neck. She was admitted to hospital at 9 p.m.

Neurological examination revealed marked engorgement of the veins in the right *fundus oculi*, with a small subhyaloid hæmorrhage below the optic disk. The deep reflexes were equal and extremely active, the abdominal reflexes were equally feeble on both sides and the plantar responses were flexor in type. There was a moderate degree of stiffness of the neck. Otherwise examination revealed no signs of significance.

By lumbar puncture 20 cubic centimetres of evenly blood-stained fluid were slowly withdrawn. The initial pressure was 240 millimetres, the final pressure 110 millimetres of cerebro-spinal fluid. The patient remarked afterwards that she felt the headache going while the doctor was "working on her back". The cerebro-spinal fluid contained 239,500 red blood cells, six lymphocytes and two polymorphonuclear cells per cubic millimetre. The supernatant fluid after centrifugalization showed a faint yellow tinge. The total protein content of the fluid was 0.085%, and there was no reaction to the Wassermann test.

There was no recurrence of headache. The temperature rose to 37.2° C. (99° F.) for four days and then remained normal. Stereo-lateral and antero-posterior skiagrams of the skull revealed no abnormality.

Two weeks later she first noticed a deep-seated pain behind the right eye, which recurred every two or three days. On July 15, 1936, she felt "giddy and weak, as though her head was full", and later in the evening she first noticed a noise, like something running and yet ringing, in her head and a creepy feeling above the right eye. She felt as though a needle were being stuck right through the centre of the eye, and later felt that the eye was filling up and would burst. She experienced nausea and desire to pass urine very frequently. The noise, which she said suggested her heart beating inside her head, was always present when she lay down, but was minimized if she lay flat on her back with her head turned to the left side, and abolished by sitting or standing. The unpleasant sensation of something crawling under the skin above the right eye became continuous. Later it spread to the temple and over the right side of the head to the vertex. On July 21, 1936, her right upper eyelid commenced to droop and she was unable to open the eye properly. A few days later she commenced to see double, and within a short time could see clearly only when the right eye was covered.

On her readmission to hospital on August 22, 1936, there was found to be complete paralysis of the third cranial nerve on the right side, indicated by ptosis of the upper eyelid, a dilated inactive pupil and her inability to elevate and adduct the eye, downward movement being also very limited. In spite of the complaint of dysæsthesia over the forehead, no alteration in sensation was detected when the region of supply of the ophthalmic division of the fifth cranial nerve was tested. Otherwise there were no abnormal physical signs.

Lumbar puncture revealed a clear fluid under an initial pressure of 190 millimetres of cerebro-spinal fluid. No cells were seen in three cubic millimetres of the fluid, which contained 30 milligrammes of protein per 100 cubic centimetres.

It was obvious that the patient had an aneurysm upon the right internal carotid artery, situated upon the intracranial part of that artery in such a position as to involve the third cranial nerve and the ophthalmic division of the fifth cranial nerve, and at a previous juncture to leak into the subarachnoid space. The pain became intolerable, and when operation was discussed with the patient, she chose any course rather than remain as she was.

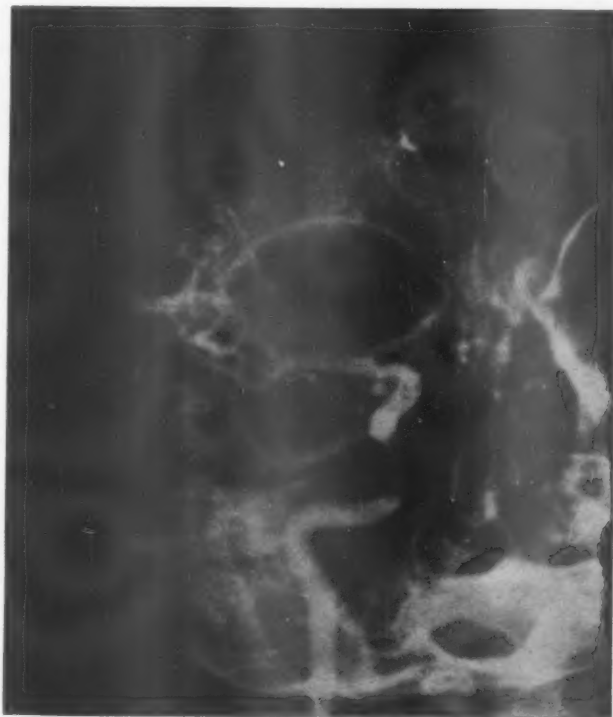


FIGURE III. Antero-posterior skiagram of the skull following injection of "Thorotrast". The internal carotid artery, anterior cerebral and middle cerebral arteries are clearly seen.

On September 2, 1936, Dr. A. E. Coates exposed the right internal carotid artery under local anæsthesia, exposure being facilitated by division of the common facial vein. With the aid of a sling of catgut, which also served to constrict the artery on the proximal side, the artery was drawn into the wound and pierced by a needle of large bore. Ten cubic centimetres of "Thorotrast" were then rapidly injected, and a skiagram of the skull in the antero-posterior position was quickly taken during the injection of the last cubic centimetre of radio-opaque substance. The X ray film was rapidly changed and another exposure made. With the head rotated to the left the procedure was repeated and lateral views were obtained. The results are shown in Figures III, IV and V, demonstrating clearly the aneurysmal sac upon the internal carotid artery just proximal to its bifurcation. In the second exposures in each position the "Thorotrast" has been swept into the veins, but still remains in the

aneurysm. The diagnosis of aneurysm upon the internal carotid artery having been confirmed, the internal carotid artery was abruptly occluded. The patient was able to perform at command voluntary movements of the left lower limb after the occlusion. However, she became drowsy and slow in responding to questions, and when her left arm was uncovered, it was found that she was unable to move it voluntarily, although the power in the lower limb remained good. Rapid examination revealed left-sided



FIGURE IV. Skiagram taken very quickly after that shown in Figure III. The aneurysmal sac is still filled with "Thorotrast", which elsewhere has been swept into the veins and venous sinuses.

lower facial weakness of moderate degree. The tongue was protruded slightly to the left. Movements of adduction, extension and, to a slight degree, abduction could be carried out at the shoulder, but were very weak. Flexion was not elicited and she was unable to make any movement at the distal joints. The muscles were flaccid. The power of the muscles of the lower extremity appeared to be unimpaired. The deep reflexes in the left upper limb were absent, while those in the lower limbs were present. The left-sided plantar response was an indefinite one, but was more flexor than extensor. She was unable to name a match-box placed in her left hand. At

this time there was a marked reduction in her mental faculties. It was noted that the arm had a bluish-red appearance. It was obvious that the circulation through the middle cerebral artery was insufficient to nourish the neurones of the grey matter of the lateral surface of the hemisphere. The collateral circulation is developed chiefly through the anterior communicating artery from the left carotid artery and through the posterior communicating artery from the basilar artery. The function of the leg was unimpaired, probably because the circulation through the anterior cerebral artery was not so much interfered with, this artery receiving blood more directly from the anterior communicating artery. The anatomical distribution of branches to the internal



FIGURE V. Lateral skiagram of the skull after injection of "Thorotrast", showing the aneurysmal sac with its origin from the internal carotid artery. The anterior cerebral, middle cerebral and posterior cerebral arteries are clearly seen.

capsule may have played a part in determining the escape of the leg fibres at this level, but the highest nervous elements, the cortical neurones, would be affected by anaemia before the efferent fibres.

It was decided to untie the ligature upon the internal carotid artery, and this was done within ten minutes of the initial ligation, the delay being due to failure to note immediately the paralysis of the arm. Almost immediately, for it seemed to coincide with the moment of release of the ligature, so rapid was the response, the patient was able to move her left arm well and its colour changed from bluish-red to white.

Examination fifteen minutes after the untying revealed a marked improvement in her mental state; she was still drowsy, but could cooperate sufficiently for a full neurological examination. She accurately gave the date, the number of her ward, her name and full address, and was approximately correct in judging the time. She could

remember returning from the X ray room to the theatre table, but thought that she had fainted just afterwards.

The visual fields were full, and there was no attention hemianopia. The fundal vessels were normal. The signs of involvement of the third cranial nerve were unaltered. The corneal reflexes were equal and active, and a pin-point was equally appreciated on both sides of the face. There was a slight degree of weakness of the lower facial muscles on the left side and the tongue was protruded slightly to the left. There was slight weakness of flexion of the elbow and wrist and of the grip of the hand on the left side. The power of the left lower limb was normal. No alteration in tone was detected. The tendon reflexes were equally active on both sides of the body, the abdominal reflexes were absent and the plantar reflexes were flexor. Sensibility to pin-prick was equal on both sides. Appreciation of passive movement of all joints was very defective in the left upper limb and was normal in the lower limb. She was unable to discriminate between single and simultaneous double contacts over the fingers of the left hand and over the foot. She handled objects clumsily in her left hand, and was able to recognize some large objects, but not small ones.

During the next hour the function of the cranial nerves, motor signs and reflexes remained as described above, and there was a progressive improvement in sensation. She correctly interpreted coarse movements of her fingers, compass-point discrimination was more accurate, and she was able to recognize the nature of objects placed in her hand with more certainty.

An hour after release of the ligature, it was decided to attempt gradual occlusion of the vessel in the belief that, if the paresis developed, it could be immediately relieved, and this was gradually effected over the space of half an hour. Progressive improvement in discriminative sensibility continued during the gradual obliteration, and fifteen minutes after the artery was tied the patient said that she felt exhausted, but not drowsy. Appreciation of single and double contacts was normal, she correctly interpreted the position of her fingers, and she was able to name small coins. The incision was then closed.

At 5.30 p.m. the patient said that the pain behind the eye and the sensation of movement above it had disappeared. She was able to open the left eye more easily. The facial weakness was less, and the only abnormal signs present were slight drooping of the outstretched left arm and hand and a very poor plantar response on the left side.

The next day the patient was well and very pleased with the complete relief from pain; but at 11 p.m., some thirty-five hours after the termination of the operation, she was found to be semicomatose, unable to speak and paralysed on the left side. Extension and adduction and, to a very slight degree, abduction of the shoulder were the only movements possible in the upper extremity. The power of dorsiflexion of the left foot was lost, but other movements were feebly present in the lower limb. The left-sided tendon reflexes were present, but diminished, the abdominal reflexes were all absent, the right plantar response was flexor in type and the left extensor. She resented pin-pricks on the right side of the body, but not on the left. The retinal arteries on the left side were normal, suggesting that since the ophthalmic artery was not involved, thrombosis of the internal carotid artery had not occurred.

Lumbar puncture revealed a slightly yellow fluid under an initial pressure exceeding 320 millimetres. After removal of 20 cubic centimetres the pressure was 170 millimetres. The fluid contained 194 red cells per cubic millimetre and 0.015% of protein, and qualitative tests for globulin were negative. She spoke a few words after the puncture, but during the night she lapsed into unconsciousness and respiration became laboured. It was considered possible that embolism or slowly spreading thrombosis had occurred in the middle cerebral artery. As, however, the previous occlusion had caused hemiparesis, it seemed possible that oedema, perhaps dependent upon the earlier ischaemia, had developed in the cerebral hemisphere to a sufficient degree to impair seriously the already depleted blood supply. When the hemiplegia was reported, probably some time after its onset, no good, and perhaps some harm, would have resulted from untying the ligature on the artery. For this reason the evacuation of a large quantity of cerebro-spinal fluid and intensive treatment by dehydration were undertaken.

On the next morning the hemiplegia was complete, with diminished tendon reflexes, and the patient was unconscious and unable to swallow. Lumbar puncture showed the pressure of the cerebro-spinal fluid to be 240 millimetres, and the fluid contained 208 red blood cells, one lymphocyte and one polymorphonuclear leucocyte per

cubic millimetre. Lumbar puncture on five successive days revealed a pressure varying between 250 and 220 millimetres. The pressure was reduced to 120 millimetres on each occasion. On September 6 the patient became more conscious and continuously demanded a cup of tea. The visual fields were full. There was complete loss of discriminative sensibility in the left hand and to a less extent over the foot, and the sensation to pin-point and touch was markedly impaired over the hand and arm and, to a lesser degree, over the left foot and leg.

On September 11 the pressure of the cerebro-spinal fluid was 130 millimetres. The patient was able to answer questions reasonably and remembered the preceding events of the day. The arm showed a gross increase of flexor tone, the lower limb of extensor tone, and for the first time voluntary movement was possible at the left hip. There was still a gross impairment of all forms of sensation in the upper limb, but the loss of superficial sensibility was diminishing. On this day she was troubled by a sensation of pins and needles in the left lower limb.

Progressive mental and physical improvement occurred, so that by October 30 she could walk with support. Examination on that date revealed an unimpaired mentality; she was able to describe the events after the operation until the evening of the following day, and after that she remembered nothing until four days later, when she became aware that she was unable to move her left arm and leg. The right pupil was still large and reacted neither to light nor to accommodation. There was ptosis of the right upper eyelid, but less than before the operation. The right eye could be adducted almost fully, upward movement was limited to a greater extent, and downward movement was but slightly limited. The left nasolabial fold was shallower than the right on voluntary movement.

The left upper limb was adducted at the shoulder and flexed at the elbow, with semiprone forearm and wrist and flexed fingers. Tone was greatly increased in the flexors and abductors of the shoulder, in the flexors of the elbow and in the flexors of the wrist and fingers. The power of voluntary flexion at the shoulder was absent, abduction was very weak, adduction was stronger and extension was of fair power. Flexion of the elbow, but no other more distal movement, was possible. In the lower limb there was moderate resistance to passive abduction and flexion at the hip joint, with a gross increase in extensor tone of clasp-knife type at the knee. Voluntary flexion of the thigh was very weak, abduction was better, and adduction and extension were still stronger. External rotation was of fair power, but internal rotation was very weak. Flexion of the knee joint was very weak, extension fair. No movement of the foot or toes was possible. There was no wasting. The tendon reflexes were greatly increased on the left side, with patellar and ankle clonus. The plantar response was strongly extensor.

She was able to walk when discharged from hospital, and although the unfortunate nature of events had been explained to her, she said that the "cure" was not as bad as the disease. One felt that her life would for ever be a burden, but when last seen she had compensated very well, and although unable to use her hand, she has learned to walk extremely well, swinging a stiff spastic lower limb from the hip.

It may have been impossible to avoid this accident, depending as it probably did upon an abnormality of the circle of Willis, but one wonders whether it might have been avoided had the usual surgical method of very gradual constriction of the internal carotid artery been adopted in the first instance. If, however, the right posterior communicating artery were a small vessel, especially if associated with a small anterior communicating artery, this accident would be almost inevitable. Fawcett and Blachford,⁽⁹⁾ in a study of 700 brains, found the right posterior communicating artery to be absent in 1.8% of cases, the left in 1.4% and both in 0.4%. These figures are lower than those of other observers; for instance, Stopford,⁽¹⁰⁾ in 150 cases, found the right-sided artery to be absent in 4%, the left in 3%.

It may be wise to use no more than 10 cubic centimetres of "Thorotrast", and the publication of a series of cases in which 75 cubic centimetres have been injected intravenously for hepato-splenography does not altogether allay uneasiness that "Thorotrast" may induce changes in the cerebral capillaries

favouring the development of œdema or of clotting. If the diagnosis of aneurysm upon the internal carotid artery can be made without recourse to injection of "Thorotrast", it is best to avoid this procedure.¹ Lateral and antero-posterior views, demanding two injections, are unnecessary. The procedure of ligation of the internal carotid artery is successful in so many cases that the method is worth general application, and the publication of a standard technique by a surgeon who has performed the operation many times would be of great assistance. Preliminary digital compression of the carotid artery, carried out intermittently for some days prior to operation, may solve the problem.

ACKNOWLEDGEMENTS.

It gives me pleasure to record my thanks to my colleagues who have allowed me to study their patients, and to the Editor of *The Medical Journal of Australia* for permission to republish Figures I and II.

REFERENCES.

- ⁽¹⁾ Osler and McCrae: "Principles and Practice of Medicine", Ninth Edition, page 380.
- ⁽²⁾ W. R. Gowers: "A Manual of Diseases of the Nervous System", First Edition, 1888, Volume II.
- ⁽³⁾ C. F. Beadles: "Aneurysms of the Larger Cerebral Arteries", *Brain*, Volume xxx, October, 1907, page 331.
- ⁽⁴⁾ F. J. Nattrass: "Aneurysm of the Carotid Artery in the Cavernous Sinus: Ligation of the Internal Carotid Artery: Recovery", *The Edinburgh Medical Journal*, Volume xxxv, January, 1928, page 30.
- ⁽⁵⁾ Egas Moniz: "*L'Angiographia Cerebrale*", 1934.
- ⁽⁶⁾ Norman M. Dott: "Intracranial Aneurysms: Cerebral Angio-Radiography: Surgical Treatment", *Transactions of the Medico-Chirurgical Society of Edinburgh*, Volume xlvii, 1932-33, page 219.
- ⁽⁷⁾ Leonard C. Lendon: "Cerebral Arteriography", *The Medical Journal of Australia*, Volume I, June 30, 1936, page 849; and personal communication.
- ⁽⁸⁾ F. Albright: "The Syndrome Produced by Aneurysm at or Near the Junction of the Internal Carotid Artery and the Circle of Willis", *Bulletin of the Johns Hopkins Hospital*, Volume xlv, April, 1929, page 215.
- ⁽⁹⁾ E. Fawcett and J. V. Blachford: "The Circle of Willis: An Examination of 700 Specimens", *The Journal of Anatomy and Physiology*, Volume xl, 1906, page 63.
- ⁽¹⁰⁾ J. S. B. Stopford: "The Arteries of the Pons and Medulla Oblongata", *The Journal of Anatomy and Physiology*, Volume I, 1936, page 131.
- ⁽¹¹⁾ D. W. C. Northfield and Dorothy S. Russell: "Thorotrast in Cerebral Arteriography", *The Lancet*, Volume ccxxxii, February 13, 1937, page 377.

¹The publication of a recent paper by Northfield and Dorothy S. Russell⁽¹¹⁾ affords clinical and histological evidence for the avoidance of "Thorotrast" in cases other than those of aneurysm.

A REVIEW OF THE ACUTE POST-OPERATIVE CIRCULATORY DISTURBANCES.

By SIR HUGH DEVINE,

Senior Surgeon, Saint Vincent's Hospital, Melbourne.

WITH the object of ascertaining the views in other countries in regard to the causation and treatment of acute post-operative circulatory disturbance, I made an investigation into recent foreign literature. Feeling that the information which I had gathered for my own use was not generally available and would be useful to readers of the journal, I have embodied it in this review, with some of my own views on post-operative circulatory disturbance. Of course, no review on this subject would be complete without reference to the enormous amount of work which has been done in England and America. English and American work, however, is accessible to Australian and New Zealand surgeons. This review will therefore mainly serve to show the views held in other countries about the causation and treatment of acute post-operative circulatory disturbances.

Acute post-operative circulatory disturbance of some degree may follow any severe abdominal operation. As a result of the operation, the circulatory mechanism has been subjected to a severe strain, the effects of which are manifested in much the same way as those resulting from very strenuous bodily effort. The circulatory mechanism is also disturbed by the effects of the injury associated with the operation, that is, by products arising from the destruction of tissue and from injury to the nervous system. This circulatory disturbance is usually more severe in operations in the upper than in the lower part of the abdomen. The reasons for this are that organs in the upper part of the abdomen are vital and have not only a richer sympathetic nerve supply, but also a freer lymphatic supply capable of carrying out rapid absorption—both conditions which do not obtain to the same extent in the lower part of the abdomen. Thus a life-endangering circulatory disturbance is more frequently a post-operative feature of operations in the upper part of the abdomen than in those of the lower part of the abdomen.

This circulatory disturbance is, by some writers, called shock, by others, collapse.

Post-operative circulatory disturbance or shock cannot be attributed to any one cause. It is the combined effect of many interacting factors set in motion by the operative disturbance. It may be closely simulated by a shock-like circulatory disturbance arising from a cardiac failure. In order that the surgeon may be able properly to treat a post-operative circulatory disturbance, he should be capable of analysing the complex picture often presented in this condition; he should understand the various mechanisms which produce the disturbance.

In considering this circulatory disturbance following severe operations, it is necessary to distinguish between (a) the patient who has actually (not hypothetically) a normal circulatory mechanism, and (b) the patient who starts out with some "crippling" of his circulation, either cardiac or peripheral. It will be obvious that a preexisting circulatory "crippling" may introduce a large element of cardiac failure into a post-operative circulatory disturbance which might be regarded as a pure post-operative shock, and, from the therapeutic standpoint, we know that the treatment of these conditions is diametrically opposite.

POST-OPERATIVE CIRCULATORY DISTURBANCE IN PATIENTS WITH A
NORMAL CIRCULATORY MECHANISM: SHOCK (SOMETIMES
CALLED COLLAPSE).

By the term shock we mean that dramatic condition, caused by some external influence, either psychic or physical, the chief characteristic of which is a more or less profound circulatory disturbance. A condition similar to post-operative shock may be induced by such agencies as trauma, toxæmia, anaphylaxis, hæmolytic and even by psychic disturbances. Shock therefore has many causes, all giving rise to a similar clinical picture: a sudden circulatory exhaustion, revealing itself by pallor, sweating, a rapid pulse, a rapidly falling blood pressure, increased respirations and apathy. Although we may not be quite sure of the actual causes of shock, we do know definitely that the conditions which occur in shock are the same whether it be caused by infection, by trauma, or by any of these means.

In these various types of shock the initial injury is always found in the walls of the capillaries. Capillary paralysis, capillary dilatation and increased porosity of the capillary walls follow; and, as the result of these changes, plasma passes from the blood stream into the tissues. Contraction of the small arteries and arterioles, caused perhaps reflexly, now follows, and the general result is a certain amount of circulatory stasis.

The outcome of all these changes is that less blood reaches the tissues and therefore less oxygen, that poisonous metabolites result from this insufficient oxidation, that these metabolites cause further poisoning of the capillary walls and further capillary dilatation. Following all these changes there is a decrease in the amount of the circulating blood, but an increase in the amount of blood in the "blood depôts"—the liver, spleen, the abdominal vessels and the extremities—and thus there is a rapid falling of the blood pressure. As a result of these changes and particularly of the fall in blood pressure and the decrease in the amount of circulating blood, there is a lessening of the systolic output, and thus an insufficient supply of blood reaches the right heart. At the same time, influences originating possibly from the contraction of the arteries, possibly through humoral channels, reach the adrenal bodies and adrenaline is poured out. The outpouring of adrenaline causes a compensating increase in blood pressure, and, for a time, helps to stem the tide; but as the blood pressure falls and the amount of circulating blood decreases, a phase of the circulation arises in which there is insufficient blood—and therefore an insufficient supply

of oxygen—to supply the heart muscle, the vital nerve centres in the medulla and the ganglionic centres of the peripheral vascular mechanism. A stage is thus reached at which these centres are exhausted and at which they can no longer transmit impulses to raise the blood pressure, and at which, therefore, a state of shock exists. This, then, is the condition that we find in shock, regardless of its causation. When we come to consider the cause of shock, the same unanimity is not found among the various observers as is found in regard to its manifestations.

The Exciting Causes of Shock.—That there are two main causes of shock cannot be doubted: two components, one a quick-acting neurogenic cause, and the other the effect of some slowly acting auto-genously produced poisonous substance.

No one can doubt the neurogenic causation of the shock which is produced when the testicle is crushed, when a strangulated bowel is being pressed back into the abdomen, when a sciatic nerve is being divided. A kick in the abdomen can produce death from shock, notwithstanding the fact that there has been no actual gross tissue injury. Injury to the sympathetic nerve endings can produce great shock: death has followed the ligation of the large bronchus, following the injury to the "terminal reticulum" of sympathetic filaments in such a vital organ as the lung (Sunder-Plassman⁽¹⁾). Shock in all these cases is neurogenically produced, and comes on immediately, that is, at the time of the operation—comes on dramatically and requires urgent treatment.

Neurogenically produced shock can be brought about psychically: death from shock has followed great terror.

All surgeons will agree that typical symptoms and signs of shock appear during or immediately following the operation, and, as these abate, they begin to appear again, coming on slowly after the first twenty-four hours—a condition sometimes referred to as secondary shock or as the erethic phase of shock (Rehn⁽²⁾) or as "chemical shock". It is not shock in the sense that it comes on dramatically and requires immediate treatment. This rather slowly developing phase of shock is regarded by all physiologists as being caused by a second important shock-producing component—a toxic substance arising as a result of the injury to tissue, a toxin which produces capillary injury and paralysis.

Symptoms similar to those of shock follow histamine poisoning; but this substance, which, however, is extremely difficult to isolate, has not been found in the blood of persons suffering from shock. A toxic substance has been found in the blood stream of experimentally shocked animals, and in the blood stream in cases of peritonitis. Toxic substances can be isolated from defibrinated blood and also from fresh blood (Freund⁽³⁾), which, when injected into animals, cause the symptoms of fatal shock. König⁽⁴⁾ has shown that small doses of crushed muscle extract injected intravenously can kill an animal, and he has also isolated from degenerated blood platelets and from broken-down products of nuclear degeneration, a substance which, when injected, produces shock. He has pointed out that both of these substances are found in the blood after extensive operations, after serious trauma and in cases of

severe burn. Injection of any of these poisonous substances will produce a falling blood pressure, a decrease in the amount of circulatory blood, an increase in the volume of blood in the lung, in the liver, in the spleen and in the extremities—in effect, a condition of shock. Switching out the nervous system by anaesthesia does not prevent or lessen the development of shock.

Thus, after operations, injury to the sympathetic and sensory nerves, in combination with the action of a toxic substance produced by injury to tissues, can so act on the peripheral vascular system as to produce the condition which we know as shock.

Recent investigations by König⁽⁵⁾ show, however, that this is not the whole story. This observer has shown that it is not the rapidly falling blood pressure which causes death. Out of ten animals which he had injected with a crushed muscle extract, five were injected with "Germanin", a substance which prevents the clotting of blood. In the animals which he had not injected with "Germanin", death occurred with the usual conditions found in fatal shock, namely, rapidly falling blood pressure, vascular dilatation, decrease in the amount of the circulating blood. But although those animals which he had injected with "Germanin" developed all the manifestations of shock, they survived. In some peculiar way the anti-clotting ferment prevented a fatal result. König concludes, therefore, that death from shock is not so much a result of falling blood pressure as of a sudden disturbance in the clotting system. König's observation also holds good in anaphylactic shock and in shock arising from other causes. The same result was obtained by Kyes and Strauser⁽⁶⁾ using "Heparin", another anti-clotting ferment. Therefore, König concludes that fatal shock is the result of poisonous substances derived from nuclear degeneration, and appears to be dependent on the influence of excess of prothrombin. If the formation of prothrombin is inhibited, no shock occurs. König suggests that it is a sudden enormous release of prothrombin which causes hæmolytic shock after transfusion of incompatible blood.

Thus we see that shock is a highly complex process which can be caused reflexly by nervous disturbance, by some circulating toxic product, and perhaps by a sudden disturbance in the blood-clotting system.

THE TREATMENT OF POST-OPERATIVE SHOCK IN A PATIENT WITH A NORMAL CIRCULATORY MECHANISM.

Neurogenic shock can to a certain extent be avoided, and therefore the best "post-operative treatment" of shock is carried out by the surgeon at the operation table, where he uses his physiological knowledge, his ingenuity and his skill to avoid producing neurogenic shock. A surgeon who is mentally and physically well equipped for the performance of abdominal operations—and, indeed, of any operation—will produce far less shock than one who is less competent.

Deep, prolonged and badly given general anaesthesia will produce "chemical shock". But a happy choice of an anaesthetist or of an anaesthetic may do much to prevent this. For example, in certain cases of carcinoma of the stomach in which great debility is present, the use of local anaesthesia will avoid a certain amount of shock. Nitrous oxide

gas, supplemented with ether used at certain stages of the operation when relaxation is required, may lead to fewer serious after-effects than if ether is given alone. Much handling of the viscera, tearing of adhesions, dragging on the omentum, pulling the intestines onto the abdominal wall and operating on them as they lie there, are all prolific causes of shock, causes which can either be greatly minimized or entirely eliminated.

It is sometimes wise to anticipate the onset of shock and to give during or at the end of the operation, before the patient leaves the operating table, an intravenous infusion of two pints of Ringer's solution with 8% to 10% of glucose. This infusion ensures that during the critical first twenty-four hours the heart muscle is not placed at a disadvantage because of an inadequate supply of blood circulating through its coronary arteries. Compensatory action of a heart well supplied with nourishment can then come into play to combat the effect of the dilatation of the peripheral vessels caused by neurogenic influences. If the heart is assisted early the peripheral circulatory mechanism, the failure of which has caused circulatory failure or shock, may quickly recover.

The Treatment of Shock.

The treatment of post-operative shock in a patient whose circulatory system can be regarded as healthy, is based on the following principles:

1. The blood vessels must be filled to compensate for the plasma loss and for the decrease in the amount of circulating blood.
2. The peripheral vascular system must be toned up, that is, contraction of the peripheral vessels must be stimulated.
3. The breathing centre must be stimulated.

The Restoration of the Loss in the Amount of Circulating Blood.—In post-operative shock, and even in that which follows in the train of toxæmia, it is not the myocardium which is affected, but the peripheral vascular system. And the loss of circulating blood, caused by this peripheral failure and resulting plasma loss, is best replaced by a transfusion of blood. As a substitute, Krogh recommends salt solution with 6% gum arabic—"gum solution". Concentrated sugar solution (30% to 40%) is perhaps the next best. "Gum solution" and strong sugar solution should not be administered in large amounts, for a plethora can result. Ordinary normal saline solution remains for a relatively short time in the circulatory system, and is toxic to the desanguinated heart in shocked subjects; but these drawbacks can in large measure be overcome by employing a continuous drip infusion of an 8% to 10% glucose in a saline solution (Tyrode or Ringer's) of approximately the same chemical composition as that of the plasma; and this experiment has shown to be non-toxic to the anæmic heart. The amount of such a solution which may be given varies according to the condition of the circulation, and may be as much as three to six litres in a day.

In severe cases of shock it may be necessary to give a massive transfusion of two pints or more of blood, which, of course, may have to be collected from two or three donors. Two precautions in regard to such transfusions are necessary in these cases. At the beginning the

transfusion must be given very slowly, in order to allow the body time to adapt itself to any slight incompatibility; that is, to take advantage of that natural faculty of "quick protection" which the body possesses—the "*Schnell-schutz*" of the German observers, the tachyphylaxis of the French. It is, of course, most essential that the donors' blood should be "cross-tested" against the recipient's, for it has been shown that in these very weak patients the slightest incompatibility may turn the scale against the patient. It is not enough to rely on blood grouping. A hurriedly given transfusion, even when donor and recipient belong to compatible groups, has precipitated the end in more than one case of severe shock. In such cases it is better to precede transfusion of blood by a saline infusion which is chemically identical with the blood and which contains its buffer salts in the proper percentage—the ordinary saline solutions are often toxic to the anæmic heart (Weichardt⁽⁷⁾). With such a solution the water used must be freed of any trace of organic matter by being distilled with permanganate of potash. The chemical composition of such a solution should be as follows: NaCl, 8.00 grammes; CaCl₂, 0.20 gramme; KCl, 0.20 gramme; MgCl₂, 0.10 gramme; NaH₂PO₄, 0.05 gramme; NaHCO₃, 1.00 gramme; H₂O, 1,000 cubic centimetres.

Unfortunately, when this solution is sterilized by heating, a precipitate forms. It must therefore be sterilized by being passed through a special filter or it may be sterilized in an autoclave in bottles in which any air is replaced by carbon dioxide.

Toning up the Peripheral Vascular System.—It has already been pointed out that in shock, however induced, it is not the heart muscle which is poisoned, as it is in diphtheria, but the peripheral vascular system. Therefore, in attempting to combat the condition, efforts should be made to restore a peripheral vascular efficiency by toning up the arteries, which in turn should ensure that an adequate supply of blood, and therefore of oxygen, will again be delivered to the right heart. Another result of an improvement in the peripheral vascular system is that the blood depôts—the liver, spleen *et cetera*—empty and thus increase the amount of circulating blood. Adrenaline has been used to tone up the peripheral vessels, but its action has been found to be too evanescent. Recently, however, a synthetic adrenaline marketed in Germany under the name of "Sympatol", and in America as "Synephrin hydrochloride" (P-methylaminoethanol-phenol HCl) or "Neosynephrin" (levo-metamethylaminoethanol-phenol), has been produced. It is claimed that the action of this synthetic adrenaline is more prolonged than that of ordinary commercial adrenaline and, in addition, that it can be given in larger doses. It has been shown that in patients with peripheral vascular insufficiency, after adequate injections of "Sympatol", there are considerable increase in venous pressure, an emptying of the blood depôts and an increase of sometimes 50% in the circulating blood (Oberdisse⁽⁸⁾). Continuous drip intravenous injection of "Sympatol" dextrose solution is said to be most efficient in the treatment of shock. König⁽⁹⁾ gives from three to six litres a day of an 8% to 10% dextrose pure solution, with six to ten cubic centimetres of "Sympatol" to each litre. He gives twenty drops a minute or more

as the condition of the circulation demands. Given in a sufficient dosage, König thinks that "Sympatol" (or "Synephrin") dextrose pure infusion fulfils all requirements in the treatment of shock. He also points out that in order that the various cardiac remedies may act on the heart, it must be adequately supplied with oxygen. He finds that "Sympatol" dextrose solution acts as a satisfactory nourishment for the myocardium even when this structure is receiving an insufficient supply of oxygen, and thus digitalis or "Cardiazol" may act on the heart muscle, even when placed at such a physiological disadvantage.

Stimulation of the Breathing Centre.—Active stimulation of the breathing centre hastens the circulation of the blood, and is therefore important in the treatment of shock. "Sympatol" and glucose infusions favourably influence the respiratory centre. After the inhalation of carbon dioxide, Brednow⁽¹⁰⁾ found an increase in the amount of the circulating blood and an expression of stored blood from the blood depôts, while Frimann-Dahl⁽¹¹⁾ demonstrated an increase in the rate of the venous stream in the saphenous vein.

The percentage or the amount of carbon dioxide inhaled should be large enough to produce hyperpnœa. The gas should be inhaled from a rubber bag and should be given intermittently. "Carbogen" (5% carbon dioxide in oxygen) is regarded by some Continental writers as not sufficiently strong to stimulate the respiratory centre in cases of severe shock. Carbon dioxide cannot act on the paralysis of a respiratory centre such as may accidentally be induced by the administration of "Evipan" or such intravenously administered narcotics, or after a badly given general anæsthetic. In such cases König and other Continental authors recommend that injections of caffeine, "Coramine", lobelin, should be given by suboccipital injections into the *cisterna magna*. Such treatment, they say, has on occasion revived an exhausted respiratory centre, even when there has been temporary cessation of the pulse. When the breathing centre is severely depressed, lobelin should be added to the intravenously injected "Sympatol" dextrose solution. Used in this way, it seems to be more efficacious than if used alone.

So far, then, we have been concerned with the disturbance of the circulation after operations in a patient who is presumed to have a healthy circulatory system. Shock, however, may occur in a patient whose circulatory mechanism is inadequate *ab initio*.

POST-OPERATIVE CIRCULATORY DISTURBANCES IN PATIENTS WITH A "CRIPPLED" CIRCULATORY MECHANISM.

A study of the literature reveals that between 10% and 15% of post-operative deaths are caused by a circulatory disturbance for which a preexisting lesion of the heart is mainly responsible. The anæsthetic and the strain of the operation bring to light the weakness in a "crippled" heart, a weakness which in most cases has been clinically concealed. Even severe heart lesions may not be recognizable by the usual clinical examination. Foged and Torben Geill⁽¹²⁾ studied a series of 428 patients in which a serious surgical operation was indicated. Of these, 351 were patients whose hearts had been carefully clinically examined in the cardiac clinic, and nothing abnormal had been found.

In 253 of these cases electrocardiography and Röntgen photography upheld the clinical findings that no heart lesion was present. In the remaining 100 of these patients electrocardiography or Röntgen photography, or both, gave an abnormal finding. In the first group the post-operative mortality from heart weakness was 1.1%; in the second group the mortality was 11.8%.

Coronary sclerosis and myocardial degeneration were the cardiac lesions which indicated a bad operative prognosis.

Every patient, especially if he is old or fat, who is about to undergo serious operation, should be cardiographically and radiographically examined. When examinations show that the patient has a heart weakness, and if this heart weakness is a coronary sclerosis or a myocardial degeneration, operations for the removal of gall-stones and the repair of hernia, and such operations which are not urgent, should not be performed. If the heart weakness is not great, then perhaps very often a cholecystostomy can be performed instead of a cholecystectomy and a gastro-enterostomy instead of a gastric resection.

Ewig⁽¹³⁾ has drawn attention to these early and almost unrecognized stages of heart failure which are an important factor in a so-called post-operative shock.

A post-operative circulatory disturbance may really be a combination of shock with a certain amount of cardiac failure resulting from a "crippled" heart, which was apparently normal when not subjected to any special strain. The diagnosis of how much a peripheral vascular failure and how much a cardiac failure plays in a post-operative circulatory disturbance is important, for the principles of treatment of cardiac failure component are diametrically opposed to those underlying the treatment of the peripheral vascular failure which is responsible for true shock.

A "crippled" heart is found in those patient with a mechanically defective, but compensated, heart. As a consequence of the strain of the operation it becomes manifest in aged patients with apparently normal hearts who have a poor circulatory reserve—an adequate circulatory reserve is a quality of youth. It is also seen in patients who have been swept by severe "storms" of toxic or infective disease, in those who are obese and who have led sedentary lives, and in those who suffer from arteriosclerosis or with a metabolic or any general disease.

In failure of the heart the output of blood into the aorta per minute becomes less and the systolic volume of each heart stroke, therefore, becomes less. The heart, then, as it were, seeks by increasing the frequency of its beat to counteract the effect of this diminished systolic output. If this extra cardiac effort does not succeed, then the blood which is returned from the periphery to the heart is not all thrown into the arterial system. These circulatory changes occur after the heart has been heavily loaded, as after an operation, when any latent heart weakness becomes manifest and a chain of events occurs. The blood lying before the left side of the heart stagnates in the lungs, and thereby causes a decrease in their vital capacity. This stagnation causes congestion in the base of the lung, a condition which can sometimes

be radiographically recognized, the lung being darker owing to increased vascular contents.

The first manifestation, therefore, of heart failure is an increase in the action of the heart. The blood pressure may not be altered; it may even remain relatively high. Cyanosis and dyspnoea may not be present, nor may any symptoms of heart weakness be obvious. Early manifestations of heart failure are: (i) increase in the venous pressure, which is shown by (a) an increase in the size of the veins in the arms and the neck, and (b) by an enlargement of the liver; (ii) urinary changes, (a) urobilinogen appears in the urine—a sign of insufficient circulation in the liver, (b) the urine may contain albumin and the specific gravity may increase, (c) the amount of urine may be small during the day, but may increase during the night. At this stage an examination of the heart will reveal little or nothing. Perhaps it may be noticed that the patient, when talking, cannot utter long sentences with a single breath.

As diagnosis in post-operative circulatory failure must primarily be concerned with the question as to whether there is a cardiac or a peripheral failure or both, it is instructive to compare the manifestations caused by peripheral vascular failure (that is, by shock) with those brought about by cardiac failure.

In shock the blood pressure is low, the skin is moist, the venous pressure low; the arm and neck veins are collapsed, the liver is impalpable; there is no nocturia, no albumin, no urobilinogen in the urine; the cheeks are sunken and the eyes deep set; the lungs are empty of blood; the patients preserve a horizontal position; there is a decrease in the amount of circulating blood, and a great decrease in the systolic output is present—all manifestations exactly the opposite to those found in heart failure.

In cardiac failure the blood pressure is usually not low, the venous pressure is high, the arm and neck veins are distended, the liver may be palpable; there are urobilinogen and albumin in the urine; the cheeks are not sunken as in shock, nor the eyes so deep set; the patients like to sit up; there is an increase in the amount of circulating blood, and not nearly so much decrease in the systolic output as in peripheral vessel failure (shock). It must, however, be understood that in some cases cardiac failure may be secondary to a vascular insufficiency, and may be the result of an insufficient amount of blood offered to the heart by the peripheral circulation, and therefore of an insufficient supply of blood to the coronary arteries causing a definite injury to the cardiac muscle.

Schmidt⁽¹⁴⁾ therefore points out that in order to recognize the true nature of an acute post-operative circulatory disturbance, it is necessary to examine simultaneously the three important circulatory components: (a) the amount of circulating blood, (b) the venous pressure, (c) the magnitude of the work of the heart.

The amount of the circulating blood can be estimated by injecting a measured quantity of a solution of Congo red and colorimetrically determining its dilution by the blood. The venous pressure can be measured exactly by the method of Moritz-Tabora or others. The

magnitude of the work of the heart is much more difficult to estimate. It is necessary to know how much blood the heart with each single stroke pumps into the arterial system; that is, in effect, how much blood in a minute is furnished by the systolic beat—the "heart-minute-volume". This can be determined by the Grollman's acetylene method. By this method, only practicable in large clinics, it is possible to recognize alterations in the heart-minute-volume and to see in its progressive and continued decrease the approach of danger, since after an operation the healthy circulatory mechanism reacts with a progressive and continued recovery of the heart-minute-volume.

TREATMENT OF CARDIAC FAILURE.

Cardiac Stimulants.—In the hope of increasing the efficiency of the heart, cardiac stimulants may sometimes be given. Caffeine, "Cardiazol", "Coramine", digitaline, strophanthin are drugs of this nature.

The Mechanical Aids to the Circulation.—There are certain natural mechanical aids to maintaining the circulation which can with advantage be used to help one which is failing. There are: (a) abdominal pressure, (b) abdominal respiration, (c) peristalsis, (d) position of the patient.

These aids to circulation are more or less lost after a severe operation, and the maintenance of an adequate circulation is thrown entirely on the heart and peripheral vessel mechanism.

In an operation of the upper part of the abdomen, abdominal respiration is lost partly because of the pain of the wound, and partly because of the position of the wound; which causes reflex inhibition of diaphragmatic movement.

Normally the peristalsis causes differences in pressure in the abdomen, which helps to provide a circulation in the abdominal organs. The pendulum movements of the intestines are also an important factor in keeping up a circulation in the splanchnic area. Absolute intestinal immobility is a big disability to an intestinal circulation. Thus, in post-operative treatment, with a view to helping a cardiac circulatory weakness, the first object is to make the abdominal respiratory movement as deep as possible. An abdominal bandage or rubber corset should encircle firmly the upper part of the abdominal wall, but should not go so far over the ribs that it will interfere with thoracic breathing. A tight-fitting bandage gives the patient a sense of security, and encourages him to breathe freely and deeply. The patient should sit up in bed and get out of the post-operative crumpled-up position as quickly as possible. Therefore, the patient should be awakened as early as possible by an "analeptic" drug (such as "Cardiazol"), for the longer the patient remains unconscious, the greater the disablement of the circulation.

Venous Congestion.—The venous congestion which follows cardiac insufficiency requires special measures. In cardiac failure combined with shock or obscured in a shock-like clinical picture, the large infusions often used in true shock would perhaps have a fatal result; for in the former condition there is an increase in the amount of the circulating blood, while in the latter there is a decrease. Venous congestion occurs

in those patients who are, in their ordinary life, bordering on cardiac decompensation, and in whom an increase in the amount of circulating blood is imminent. These patients feel well after the operation until the amount of blood in the quickened circulation is inadequate to maintain the oxygen transport. According to Schmidt, in these patients with a bad circulation the heaping up of the metabolic products embarrasses the liver and kidneys. The importance of this is evident when we think that in addition, before the operation, there is generally some liver insufficiency. It is thus obvious that the work of the circulation is made still more difficult. In such a condition the blood pressure sinks and the pulse frequency increases. Sometimes in these cases, Ewig points out, we may be able to succour the circulation until the venous return mechanism, by the aid of its mechanical helps, again comes into action. A small dextrose infusion may tide the patient over the crisis. It may increase the loading for the heart, but the dextrose in it acts at the same time as a cardiac stimulant. It also causes a slight increase in the blood pressure, and this has a good effect on the circulation, especially on the coronary circulation. Thus the contact between the heart and the periphery may be again partially restored, and the heart is given a chance to increase the venous return into the auricle. Digitalis therapy should then be carried out.

REFERENCES.

- ⁽¹⁾ Sunder-Plassmann: *Archiv für klinische Chirurgie*, Volume clxxvii, 1933, page 25. Quoted by W. König: *Zentralblatt für Chirurgie*, November 30, 1935, page 2862.
- ⁽²⁾ Rehn: *Archiv für klinische Chirurgie*, Volume clxxvii, 1933, page 360. Quoted by W. König: *Zentralblatt für Chirurgie*, November 30, 1935, page 2862.
- ⁽³⁾ Freund: *Archiv für experimentelle Pathologie*, Volume lxxxviii, 1920, page 39. Quoted by W. König: *Zentralblatt für Chirurgie*, November 30, 1935, page 2862.
- ⁽⁴⁾ W. König: *Archiv für klinische Chirurgie*, Volume clxxi, 1932, page 447; *Der Chirurg*, January 15, 1934, page 41. Quoted in *Zentralblatt für Chirurgie*, November 30, 1935, page 2862.
- ⁽⁵⁾ W. König: *Der Chirurg*, January 15, 1934, page 41. Quoted in *Zentralblatt für Chirurgie*, November 30, 1935, page 2862.
- ⁽⁶⁾ Kyes and Strauser: *Journal of Immunology*, Volume xii, 1926, page 419. Quoted by W. König: *Zentralblatt für Chirurgie*, November 30, 1935, page 2862.
- ⁽⁷⁾ W. Weichardt: "Sterile Infusionsflüssigkeit", *Der Chirurg*, November 1, 1932, page 839.
- ⁽⁸⁾ Oberdisse: *Verhandlungen der deutschen Gesellschaft für innere Medizin*, 1934. Quoted by W. König: *Zentralblatt für Chirurgie*, November 30, 1935, page 2862.
- ⁽⁹⁾ W. König: *Archiv für experimentelle Pathologie*, Volume cxxvii, 1927, page 349. Quoted in *Zentralblatt für Chirurgie*, November 30, 1935, page 2862.
- ⁽¹⁰⁾ Brednow: *Zentralblatt für experimentelle Medizin*, Volume lxxiii, 1930, page 557. Quoted by W. König: *Zentralblatt für Chirurgie*, November 30, 1935, page 2862.
- ⁽¹¹⁾ Frimann-Dahl: "Postoperative Röntgenuntersuchungen", 1935. Quoted by W. König: *Zentralblatt für Chirurgie*, November 30, 1935, page 2862.
- ⁽¹²⁾ Jens Foged and Torben Geill: "Die prognostische Bedeutung von präoperativer elektrokardiographische und Röntgenuntersuchung des Herzens", *Acta Chirurgica Scandinavica*, Volume lxxix, December 9, 1936.
- ⁽¹³⁾ Ewig and Klotz: *Deutsche Zeitschrift für Chirurgie*, Volume ccxxxv, page 681. Quoted by W. König: *Zentralblatt für Chirurgie*, November 30, 1935, page 2862.
- ⁽¹⁴⁾ K. H. Schmidt: "Praktische Diagnostik, Prophylaxe und Therapie der postoperativen Kreislaufstörungen", *Der Chirurg*, November 15, 1933, page 865.

THE SURGICAL TREATMENT OF PROSTATIC OBSTRUCTION.¹

A NINE-YEAR REVIEW FROM THE NEWCASTLE HOSPITAL.

By THOMAS HAMILTON,
Newcastle.

IN 1937, impressed by the splendid reports from the capital cities of Australia on the lowered mortality resulting from improvements in operative technique and in the after-care of patients submitted to prostatic resection, I wondered whether the progress also applied to hospitals outside the large metropolitan areas, or only reflected the high efficiency that has been reached during the past decade by a small number of skilled genito-urinary surgeons in their respective clinics. If the following study throws any enlightenment on this question, it will have justified itself.

Every care has been taken with the figures, and cases with puzzling or doubtful records, fortunately few, have been rejected. Newcastle Hospital is particularly suited to such a study, for it is one of the largest general hospitals (300 beds) outside the State capital. It is equipped on first-class lines and is recognized by the Royal College of Surgeons of England as a training hospital. It also has the approval of our own College and the American College of Surgeons. It serves an industrial, seafaring and agricultural population of some 200,000 people. A nine-year period only is covered, as prior to 1928 the case records were not considered sufficiently satisfactory to permit of a thorough follow-up. The records available for the five-year period November, 1922, to November, 1927, however, show that twenty-two operations for removal of the prostate were undertaken with six deaths, giving a mortality of 27.27%. In twenty of the twenty-two cases operation was performed by the suprapubic route. The deaths are recorded as being due to uræmia; and five of the patients discharged as "cured" subsequently returned with suprapubic fistulæ and one with epididymitis.

These disturbing mortality and morbidity rates were not alone the prerogative of Newcastle Hospital. J. W. S. Laidley⁽¹⁾ states that in 1926 the death rate at the Royal Prince Alfred Hospital, Sydney, from suprapubic prostatectomy was in the region of 25%, and had stood at that figure for years. The death rate was not entirely the fault of the operative technique employed, for Laidley further states:

When the after-care of the patients was placed in the hands of a suitable individual, thoroughly trained in the principles of asepsis and antisepsis, an immediate result was achieved and the death rate fell to about 5%, and has been in that vicinity ever since.

¹ This paper was delivered on the occasion of the symposium on prostatic obstruction held during the eleventh annual meeting of the Royal Australasian College of Surgeons, Sydney, March, 1938.

The same intensive organization of after-care was not found possible of practical fulfilment at the Newcastle Hospital, mainly for administrative and financial reasons, and the four general surgeons handling prostatectomy work were forced to meet the situation by giving much closer personal supervision to each case, aided by the medical superintendent and the house surgeons. Encouraged, however, by the published work of Hugh Young, of Baltimore, on the perineal operation, wherein the low mortality rate of 2.8% in 2,300 cases was revealed, they abandoned the suprapubic operation in favour of the perineal, except in cases where preliminary cystoscopy and a careful clinical examination clearly indicated its use.

The following case briefly illustrates this point:

H.K., a pensioner, aged seventy-three years, was admitted to hospital on September 13, 1933, suffering from inability to void urine and showing signs of incipient uræmia. A number eight rubber catheter was introduced after some difficulty and left *in situ*. *Per rectum* the right and left prostatic lobes were not unduly enlarged or hard. His condition improved rapidly, permitting of a thorough urological investigation on the eighteenth day. His renal response to an intravenous injection of four cubic centimetres of 0.4% indigo-carmin was normal, as seen by the cystoscope. Cystoscopy also revealed a greatly enlarged and semi-pedunculated median lobe of the prostate, which extended into the cavity of the bladder and was obviously blocking the internal urinary meatus. As subsequent blood urea readings brought him within the limits of operability, a one-stage suprapubic prostatectomy was performed on the twenty-fifth day. He made a slow but complete recovery, survived another attack of uræmia, and was discharged from hospital on the seventy-eighth day. Two years afterwards he reported as having kept in good health and having full urinary control.

I feel that in this case the suprapubic route was the obvious choice in view of the pre-operative clinical findings.

The results of the change from a haphazard technique to the more careful routine, coupled with the standard perineal operation, are shown in Table I, which reviews the cases over the past nine years. It will be noted

TABLE I.
Newcastle Hospital.
Prostatic Operations.
February 9, 1928, to March 5, 1937.

Total number of operations	68
Total number of deaths	13
Total mortality percentage	19.11
Suprapubic operations	14
Deaths	5
Percentage	35.71
Perineal operations	54
Deaths	8
Percentage	14.81
Readmissions (all types) with fistula, obstruction or incontinence	10 (14.7%)
After suprapubic operation: 3 cases.		
After perineal operation: 7 cases.		
Number of cases with carcinoma (diagnosed microscopically)	6
Cancer percentage, with microscope examination (36 examinations)	16.6
Percentage (clinical)	8.82
Average number of days in hospital (post-operative)	42.35
Greatest number of days (post-operative)	148
Least number of days (post-operative)	19
Average number of pre-operative days	25.48
Average age of patients	66.22 years
Oldest patient aged	87 years
Youngest patient aged	49 years

Total Group Mortality.

Age.	Operations.	Deaths.	Percentage.
80 to 87 years	3	3	100
70 to 79 years	22	5	22.7
60 to 69 years	30	2	6.6
50 to 59 years	12	3	25.0
40 to 49 years	1	0	0

Perineal Group Mortality.

Age.	Operations.	Deaths.	Percentage.
80 to 87 years	3	3	100
70 to 79 years	16	2	12.5
60 to 69 years	23	1	4.3
50 to 59 years	11	2	18.1
40 to 49 years	1	0	0

that the total hospital mortality rate fell from 27% to 19%, and that in the perineal group the mortality was much better still, being 14.8%. In the largest age group, 60 to 69 years, 23 patients were operated on with only one death, a mortality rate of 4.3%.

We are not proud of the rate as a whole. By comparison with that obtaining in metropolitan hospitals we are rather ashamed of it. Our statistics for private patients, on the other hand, are much better, S. S. Gardiner,⁽²⁾ in 78 perineal prostatectomies, carried out over a period of fifteen years, reporting a mortality rate of under 5%.

Nor are we proud of our morbidity rate, which, over a period of four or five years, although figures are not always an index of the true position, is in the vicinity of 58%, an appalling figure (Table II). In Table I the more recent morbidity rate, as gained from readmission records only, is 14.7%.

Of 45 patients who, after prostatectomy, were discharged from the hospital as "cured", replies to follow-up letters inquiring after their progress were received from 33. Fourteen wrote that they were well, grateful for the operation, and without urinary symptoms. Four replied that they were well, but had slight urinary incontinence. Seven replied that they were in poor

TABLE II.

*Nine-year Series, Newcastle Hospital.**Follow-up of 43 Patients Discharged as "Cured" after Prostatectomy.*

Number of letters answered	33
Perineal operation	29
Suprapubic operation	4

Results.

Good (no urinary symptoms)	14
Fair (slight urinary symptoms)	4
Poor (sinuses and incontinence)	7
Died (including 4 cases within one year)	8

health, and had unhealed sinuses and no bladder control. Eight were dead, four having died within one year of discharge from hospital. Although the series is not a large one, it reveals a very unsatisfactory and alarming morbidity.

We are convinced, from our own experience and from that of the Royal Prince Alfred Hospital,⁽¹⁾ that the main essential in further improving the mortality and morbidity rates is to organize much more intensively the after-care of the patient in the public wards. Newcastle Hospital has a loyal and willing nursing staff, but even this fact does not counteract the difficulties of frequent staff changes and the absences on week-ends and holidays of those engaged in prostatectomy nursing. The late R. K. Lee-Brown⁽³⁾ pointed out that if ever a surgeon was at the mercy of a hospital staff, it was the urologist following a prostatectomy with primary closure. He further emphasized that the first and more essential requirement was a reliable staff that appreciated the meaning of asepsis, and was willing to give continuous and faithful attention to the strictly aseptic after-treatment of the patient. On the other hand, the late S. Harry Harris⁽⁴⁾ claimed for his technique that it was characterized by a very easy convalescence, and that the patients required on the average far less individual, albeit skilled, attention after operation than those submitted to everyday methods of prostatectomy. His very low mortality rate of 2.7% in 469 cases is excellent and tallies closely with the 2.8% of Hugh Young,⁽⁵⁾ the leading exponent of the perineal operation. It would be interesting to ascertain the morbidity figures in these series and also, in order to evaluate them properly, the "bad risks" which were included.

Australian surgeons⁽¹⁾ have stated that the perineal operation is technically more difficult than the suprapubic operation and needs specialized instruments. In Newcastle we have not found this to be so. Apart from a pair of Young retractors and the use of Young's prostatic retractor, which is comparatively easy of manipulation, the ordinary surgical armamentarium only is required. A knowledge of the perineal surgical anatomy is essential. The equipment needed stands out in marked contrast with the specially designed boomerang needle-holders and the electrically lighted bladder retractors required in the modern suprapubic operation.⁽⁶⁾ However, the respective merits of the two operations will always be a subject for discussion, and I will content myself with drawing attention to the fact that in this series the use of the perineal operation in the hands of general surgeons has definitely produced a lower mortality rate, as compared with that which had followed suprapubic operation in the same hospital.

It may be argued that a surgeon has no business undertaking a prostatectomy unless he is satisfied that meticulous after-care can be given to the patient by a specially trained staff, but in busy public hospitals outside the main training schools this is not always practicable. Many patients refuse to be sent elsewhere and demand treatment. Then, too, in the case of derelict patients of poor physique and of the public hospital type, the surgeon is often confronted with the problem: "Shall I take the risk of giving the patient a slender chance by operation or shall I let him drift and eventually die from a catheter pyelonephritis?" Now that transurethral resection is becoming established as an effective technique, it may help to solve this problem in the future.⁽⁷⁾

Sixteen years ago, as a house surgeon, I saw a man actually sustain a rupture of the bladder from over-distension while awaiting his turn to have sounds passed in the out-patient department. One likes to think that such days are past; but in Australian hospitals, with the exception of those quoted above which have, under the stimulus of the fine work done by the late Harry Harris, materially improved their prostatic work, the challenge of an over-high mortality rate is still with us. It can and should be met. The Royal Australasian College of Surgeons and the writers^{(8) (9) (10) (11)} quoted in the bibliography have pointed the way.

CONCLUSIONS.

1. An attempt has been made to show the status of prostatic surgery in a large general hospital over the past nine years. It is claimed, not without sorrow, that the situation revealed is probably an accurate index of the results obtained in similar work in areas outside the highly specialized metropolitan centres.

2. The chief factors calling for improvement are not so much the surgical technique or the method of approach, but the after-care of the patient and the more conservative treatment of the "bad risks".

3. Beyond that, a lowered mortality in a public hospital where a large proportion of prostatic cases are "bad risks" seems only possible by the increased use of the method of transurethral resection in the hands of a highly skilled operator.

4. It is interesting to note that the cancer percentage of 16.6 in the series (Table I) is closely akin to the figures recently published by H. H. Young for the United States of America.⁽¹²⁾

REFERENCES.

- ⁽¹⁾ J. W. S. Laidley: "A Survey of Prostatic Surgery Today", THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY, Volume vi, Number 1, July, 1936, page 54.
- ⁽²⁾ S. S. Gardiner: Personal communication.
- ⁽³⁾ R. K. Lee-Brown: "Primary Closure in Prostatectomy", THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY, Volume ii, Number 4, April, 1933, page 339.
- ⁽⁴⁾ S. Harry Harris: "Suprapubic Prostatectomy with Closure", THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY, Volume iv, Number 3, January, 1935, page 226.
- ⁽⁵⁾ Hugh H. Young: "Young's Practice of Urology", Philadelphia, March, 1926.
- ⁽⁶⁾ H. B. Devine: "Prostatic Technique", THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY, Volume i, Number 4, March, 1932, page 421.
- ⁽⁷⁾ H. C. Bumpus, junior: "Transurethral Resection: Its Indications, Limitations and Complications", *Surgery, Gynecology and Obstetrics*, Volume lxiv, Number 2A, February 15, 1937, page 324.
- ⁽⁸⁾ E. R. Reay: "The Surgery of Prostatic Obstruction", THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY, Volume iv, Number 2, October, 1934, page 130.
- ⁽⁹⁾ Reginald Bridge: "Operations of the Urinary Tract under Local Analgesia, with Particular Reference to Prostatectomy", THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY, Volume ii, Number 2, October, 1932, page 190.
- ⁽¹⁰⁾ Chas. M. Greenslade: "Sidelights on Disease of the Prostate Gland", THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY, Volume vii, Number 2, October, 1937, page 127.
- ⁽¹¹⁾ R. J. Silvertown: "Sutural Hæmostasis in Suprapubic Prostatectomy", THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY, Volume iii, Number 3, January, 1934, page 276.
- ⁽¹²⁾ Hugh H. Young: "The Radical Cure of Cancer of the Prostate", *Surgery, Gynecology and Obstetrics*, Volume lxiv, Number 2A, February 15, 1937, page 472.

TRANSURETHRAL PROSTATIC RESECTION.

THE INDICATIONS FOR ITS PERFORMANCE AND ITS RESULTS.¹

By M. S. S. EARLAM.

[From the Department of Urology, Royal Prince Alfred Hospital, Sydney.]

THE question of when should resectoscopic treatment be carried out in the treatment of prostatic obstruction is one that is in great part bound up with the other subject that I am to discuss, namely, the results of resectoscopic treatment, and I have therefore thought it preferable to discuss these two aspects of prostatic obstruction more or less together, passing from one to the other and back again as occasion arises.

In the first place, then, before starting to discuss the indications for the operation at all, I should like to mention briefly one aspect of its results, which I know is far from being generally accepted or appreciated, and that is, that provided that the surgeon has the necessary experience and technical skill, it is possible by means of transurethral resection to achieve an immediate symptomatic cure, with corresponding complete satisfaction to the patient, of a prostatic obstruction, not only of any pathological nature, but also of practically any size. It is, therefore, speaking generally, not correct to say that some cases of enlarged prostate may be resected while others should not, or that a middle lobe is suitable for resection while lateral lobe enlargement is not. The suitability in most cases depends on the attitude and technical qualifications of the surgeon and nothing else. Few, however, have the skill, the patience or the inclination to make resection a routine, so that the question that those of us who practise resection have to decide, in the absence of a definite indication for it, is whether, in our hands, the patient is likely to be better off with a resection or a prostatectomy, and whether or not in a given case the rather greater expectation of complete and permanent cure that should follow a successful prostatectomy is offset by the greater risk with which it is associated.

So far as the indications for transurethral resection are concerned, there are, I think, two groups of patients: one where the indication is absolute and open operation is contraindicated, and a second where the indication is relative, that is to say, where one surgeon may carry out a perfectly justifiable prostatectomy while another, particularly in cases where the risk, though greater than usual, is not sufficient to preclude prostatectomy, may prefer the safer operation of transurethral resection. It is, therefore, not possible to give a list of clear-cut indications for transurethral resection. What I propose to do, after shortly reviewing my own figures, is to mention briefly

¹ Delivered on the occasion of the symposium on prostatic obstruction, held on March 24, 1938, during the eleventh annual general meeting.

those conditions in which I consider that the indication for transurethral resection is definite and open operation contraindicated, and, for the rest, which constitute the bulk of the material that one sees, to discuss the question of permanence of cure after resection, with particular reference to several patients in my own series.

The three principal types of obstruction with which one has to deal are median bar, carcinoma and simple hypertrophy.

1. MEDIAN BAR.

The small fibrous prostate should not be treated by open surgery, and furnishes a definite indication for transurethral resection. The operation takes about ten minutes, there are usually no technical difficulties, the stay in hospital is short, and the results, with proper selection of cases, are excellent. In the same category, while pathologically distinct, can be placed early or moderate subcervical lobe enlargement without lateral lobe hypertrophy.

Table I shows the results in my own series of this category of patients.

TABLE I.

Number of patients	35
Repeat resection	1
Satisfactory results	32
Unimproved	3
Tabetic bladder	1
Interstitial cystitis	1
Small bladder with multiple diverticula	1
Late pathology	1
Developed hæmaturia due to subsequent lateral lobe enlargement.	1
Mortality	Nil

2. CARCINOMA.

Carcinoma of the prostate, once clinically diagnosable, is to all intents and purposes incurable, and provided that the carcinoma is not too extensive when the patient is first seen, transurethral resection is generally accepted as being the procedure of election, and will often dispense with, and at other times defer, the otherwise inevitable cystostomy. If the carcinoma is very extensive when the patient is first seen, it may be extremely difficult to make the patient comfortable for any length of time after the extensive resection that one has to do, and in these cases I have at times preferred to carry out cystostomy without further ado. Transurethral resection, like cystostomy, should be deferred until the patient's symptoms demand surgical interference.

I have had fifteen patients with carcinoma. Cystostomy was carried out in two and resection in the remaining thirteen (see Table II). In one case, number 8 in the series, extension of the disease appeared to be accelerated by the resection.

His prostate was not large and his history extended over five years. Nine months after resection he developed retention and requested cystostomy, and died about nine months later, aged seventy.

In the remaining twelve the results, though temporary, were quite reasonably satisfactory.

TABLE II.

No.	Age.	History.	Date 1st T.U.R.	$\frac{3}{12}$	$\frac{6}{12}$	$\frac{9}{12}$	$\frac{12}{12}$	$\frac{15}{12}$	$\frac{18}{12}$	$\frac{24}{12}$	$\frac{36}{12}$
1	75	Ret.	31/1/35	—	Ret. Cath.	Ret. T.U.R.	—	—	Ret. T.U.R.	—	Inc Cyst.
2	72	Diff.	18/2/35	No further report.							
3	82	Ret.	9/5/35	—	—	—	Ret. T.U.R.	—	—	D.	
4	58	Ret.	13/5/35	No further report.							
5	54	Ret.	13/5/35	—	Ret. T.U.R.	—	—	—	—	D.	
6	82	Ret.	10/6/35	No further report.							
7	81	Ret.	24/6/35	No further report.							
8	69	Diff.	4/8/35	—	—	Ret. Cyst.	—	—	D.		
9	60	Ret.	2/9/35	—	D.						
10	78	Ret.	19/9/35 (2)	—	—	—	—	Ret. Cyst.	D.		
11	79	Ret.	14/12/35	—	Ret. Cyst.	—	—	—	—	—	
12	83	Ret.	3/2/36 (2)	No further report.							
13	53	Ret.	25/9/37	—	—	—	—	Well.			

Ret. = Retention.
Inc. = Incontinence.
Diff. = Difficulty.
Cath. = Catheterized.

T.U.R. = Transurethral resection.
Cyst. = Cystostomy.
D. = Died.
(2) = Two resections necessary.

3. SIMPLE HYPERTROPHY.

I have somewhat arbitrarily divided the patients of this largest and most contentious group, more or less roughly according to the size of the prostate, and in particular of the lateral lobes, into three grades, I, II and III. In grade I the amount of lateral lobe enlargement may be described as slight, and in some cases accompanied, in other cases not, by middle lobe enlargement. In grade II the lateral lobes are moderately enlarged, again with or without the presence of a middle lobe, while in grade III the lateral lobes are large. I shall first briefly give the figures for these three groups.

In group I (Table III) are 36 patients, of whom two required a second resection. In three, to my knowledge, the result was unsatisfactory; in one due to the removal of insufficient tissue, while the second had a neurogenic bladder and it was hoped that the prostate might at least in part be

TABLE III.
Hypertrophied Prostate.

Grade I.		
Total number of patients	..	36
Repeat resections	..	2
Unsatisfactory results	..	3
Insufficient tissue removed	..	1
Neurogenic bladder	..	1
Persistent frequency with clear urine	..	1
Late pathology	..	2
Hematuria due to lateral lobes not treated at first resection	..	1
Recurrence of hypertrophy	..	1
Mortality	..	Nil

responsible for his symptoms, and the third finished with persistent frequency, with a clear urine, for which I am still unable to do anything. The rest had a perfectly satisfactory immediate result. One or two I have seen as long as four years afterwards still perfectly symptomless, and others at shorter periods, while quite a number have not reported back as instructed. Two came back at three and a half and three years respectively, the first complaining of difficulty, the second of hæmaturia; I shall speak in more detail of these at a later stage. There were no prostatectomies in this group of patients.

In group II (Table IV) were 55 patients who underwent resection and twelve prostatectomies. Nine patients required two resections. In five

TABLE IV.
Hypertrophied Prostate.

Grade II.	
Total number of patients	55
Repeat resections	9
Unsatisfactory results	5
Persistent retention (also had cerebral arteriosclerosis)—	
cystostomy	1
Persistent frequency with infection	3
Persistent frequency with clear urine	1
Late pathology: Hæmaturia—prostate increased in size	2
Mortality	2
Suprapubic prostatectomy	12

patients the result to my knowledge was unsatisfactory. One still had complete retention after two resections and had cystostomy carried out under local anæsthesia. He had general and cerebral arterio-sclerosis, and the physician who saw him in consultation agreed that he was far from fit enough for prostatectomy. Three patients had persistent frequency and pyuria, almost certainly due to insufficient removal of tissue, and one had persistent frequency, with a clear urine. Two patients died following operation.

To one, aged seventy-five years, prostatectomy had been refused elsewhere four years previously. He had advanced myocarditis and demanded operation at whatever risk. He had slight bleeding on the fifth post-operative day, requiring frequent irrigation, of which he was most intolerant. From then on he refused food and would take little fluid, developed incontinence of fæces and died on the seventeenth day.

The second, aged seventy-one years, also had advanced myocarditis, with glycosuria. In his case the machine was not cutting well, and I was not at all satisfied with the amount of tissue I was able to remove; but after his death three weeks after operation, apparently from pyelonephritis, he was found to have a large pelvic abscess.

Two patients of this group returned three years later with hæmaturia, and of the rest some are known to be perfectly well, a few have died from other causes, and the rest have not reported back.

In group III (Table V) are sixteen patients subjected to resection, and nine of these had to be resected a second time. There was no mortality and

TABLE V.
Hypertrophied Prostate.

Grade III.	
Total number of patients	16
Repeat resections	9
Unsatisfactory results	—
Late pathology—urethral stricture	1
Mortality	Nil
Suprapubic prostatectomy	19

no result that could be classed as unsatisfactory, with the exception of that in one patient who developed a traumatic stricture that requires periodic dilatation. In this group also were nineteen prostatectomies.

UNTOWARD RESULTS OF TRANSURETHRAL RESECTION AND CONTRA-INDICATIONS.

1. Mortality.

In my own series the mortality can be tabulated as follows:

TABLE VI.

	Median Bar.	Carcinoma.	Hypertrophy.			Total.
			Grade I.	Grade II.	Grade III.	
Patients ..	35	13	26	55	16	145
Resections ..	37	19	29	65	25	175
Mortality ..	—	—	—	2	—	2

This gives a mortality of 1.4% of patients and 1.15% of resections, and as both patients who died were, as has been mentioned, quite unfit for prostatectomy the mortality in reasonably good surgical risks was nil.

2. Complications of Operation.

Hæmorrhage was not a prominent feature of the series. Undoubtedly it was troublesome at times, sometimes extremely so, but no patient in the series required cystotomy for clot retention or to arrest bleeding, and no patient had to have the resectoscope passed a second time to stop bleeding after his return to the ward.

Sepsis has at times, as ever in prostatic surgery, been a worry. Several patients had pyelonephritis in severe form; one, in fact, after repeated rigors, developed toxic jaundice, then for some reason made a surprising recovery.

Post-operative stricture has occurred in a few instances, due presumably to the large calibre of the instrument used. In three cases that I remember it has caused retention of urine and required filiform dilatation. In one other case a pin-point stricture developed at the external urinary meatus.

I have had two cases of partial incontinence of urine. These were numbers 131 and 133 in the series, and I do not think that they were due to cutting of the external sphincter, but more likely to the current jumping from the cutting loop on to the metal sheath that I was using at the time, and passing thence through the external sphincter with which the sheath is in contact to the indifferent electrode under the patient's buttocks. Since I have reverted to the bakelite sheath I have had no further trouble of this nature.

3. Contra-Indications.

Before passing to a general consideration of the indications for and results of transurethral resection, I should mention one or two contra-indications. Apart from the size of the enlargement, a varying criterion among different surgeons, the principal contraindication is a small urethra, as post-operative stricture may occur and be most troublesome. Calculus is

not usually a contraindication, though one stone that I was quite unable to crush proved to be. One consideration, however, should be mentioned, and that is the fact that some apparently simple prostates on removal prove to have small malignant foci within, and may be cured by prostatectomy, whereas, of course, with transurethral resection recurrence is practically certain. This superficially is an argument in favour of suprapubic prostatectomy in all cases, but figures at the Royal Prince Alfred Hospital, of which I have been through the records, do not support it.

I have been able to find records of 267 prostatic enlargements that were clinically simple, were shelled out easily and were examined histologically (Table VII). Three of these were extensively malignant, of whom one patient

TABLE VII.

Total apparently simple prostates examined histologically	267
Extensive malignancy (one returned later—inoperable)	3
Early malignancy	2
Doubtful	8
Total possible cures of malignancy by prostatectomy	12 = 4.5%
Mortality following prostatectomy (five years ending December, 1937) ..	= 7.4%
Mortality following resection	= 1.4%

returned later with a frank malignancy, in two there was early carcinoma, and in eight cases the pathologist was doubtful as to whether the prostate was carcinomatous or not, the report in most cases suggesting a possible pre-malignant condition. This gives a maximum total of 12 cases out of the 267 who might conceivably be cured of carcinoma by prostatectomy, or 4.5%.

Over the last five years the mortality following suprapubic prostatectomy in the public wards of this hospital has been 7.4%, a figure which represents the combined mortality of general surgeons and urologists, while, as I have pointed out, the mortality following transurethral resection in this series is 1.4%, while in patients fit for prostatectomy the mortality is nil. The records, then, of this hospital, admittedly of course a small series, do suggest that in the average series of 100 prostatics treated by resection, the fact that by doing prostatectomy we could possibly have saved four or five patients from dying of carcinoma by killing off six or seven of their fellows is not a very cogent argument for doing prostatectomy in all cases.

INDICATIONS AND RESULTS IN GENERAL.

Passing now to a more general discussion of the place of transurethral resection in the treatment of simple prostatic hypertrophy, I should like to repeat my previous statement that a well-executed resection will give a completely satisfactory immediate result in the great majority of cases of enlarged prostate. To discuss the results of an imperfectly executed operation serves no useful purpose, and I shall not attempt it. I should like, however, to mention one important point which has a bearing on the results. If the prostate is of any size, grade II or larger, for the result to be satisfactory the resection must be extensive, and the more extensive the resection, the less is the post-operative morbidity and the quicker is the patient's reversion to normal micturition. Something more than the so-called canalization of the prostatic urethra is called for. The resection must extend right around the vesical neck, all intravesical prostatic tissue must be removed, and as much tissue as possible must be removed from the lateral lobes.

The question which is still contentious is the duration of the patient's symptomatic relief. In very many cases this is quite sufficiently permanent, and it appears perfectly justifiable to assume that if a patient has had an efficient resection and a satisfactory immediate result, he can almost always expect a five-year cure at least, while if all the hypertrophied tissue be removed, as can with increasing experience be more and more often achieved, the result should be as permanent as that following suprapubic prostatectomy. I have several four-year cures after resections that I would not now class as completely efficient, and in which much adenomatous tissue was left behind. If, therefore, the patient has a short expectation of life and requires operation, I think resection is definitely preferable if it can be carried out with good prospects of success.

Apart, then, from the two conditions in which open operation is contra-indicated, namely, median bar and carcinoma, there is a wide range for the employment of resection, by the experienced surgeon, in the treatment of simple hypertrophy. In the small adenomatous prostate that requires operation an excellent result with practically no risk is almost certain, and in this condition I consider transurethral resection the treatment of election, whatever be the age and general condition of the patient. Although one certainly cannot assure a young patient that a resection will save him from future prostatectomy, one can, if the resection has been complete, state with truth that his prospects of permanent cure are good.

In larger prostates resection is almost invaluable in the treatment of the poor surgical risk, not perhaps so strikingly in the case of the patient with a poor renal function, on whom, after adequate preparation, prostatectomy can usually be safely undertaken, but more so in the patient who is rendered a poor or impossible risk for prostatectomy by pathology outside his urinary tract. Several patients in the present series developed complete retention of urine simultaneously with a cerebral hæmorrhage or a coronary occlusion, while one had arterio-sclerosis with early congestive cardiac failure. Each was unable to pass urine on the several occasions on which his catheter was removed, and each had a transurethral resection under low spinal anaesthesia with a completely successful outcome. In this type of patient, however, a cystostomy is far preferable to and much safer than a badly done resection. I also consider resection the operation of election if there is any suspicion of carcinoma.

For the rest, in the treatment of the patient who is a reasonably fit subject for prostatectomy, the indications for resection are the same as those for prostatectomy, that is, the operation must be (a) one of surgical necessity, in cases where the patient cannot carry on as he is, or is becoming progressively worse, or (b), one of surgical advisability, where urinary obstruction is present in any degree. In the earlier cases, when the symptoms are mild and not progressive and the patient's general condition is good, the operation is very definitely one of election; and here the patient's symptoms should be of such degree as to justify the performance of an operation associated with a definite, if low, mortality. In this category the only difference between the indications for open and transurethral operation is that one can tell the patient that if he desires surgical relief it can be carried out by means of resection with short hospitalization and minimal risk. One point, however, that I am convinced is of great importance in the

application not only of resection, but also of prostatectomy, is that where surgery is a matter of election, the symptoms must be of sufficient degree to make the surgeon confident that operation will be followed by an adequate degree of improvement, or, in other words, that the patient will really feel better after operation. If the complaint is one of hesitancy or difficulty and a poor stream, one can promise this with full confidence, but if the patient's only complaint is one of mild frequency, the situation is totally different. I have seen more than one such patient rendered infinitely worse by prostatectomy, and, in the same way, I think any surgeon who would promise such a patient relief by resection is looking for trouble. In this class of patient surgery in my opinion is not even a matter of election; I consider it definitely contraindicated. While on this subject it may be mentioned, though it is now hardly necessary, that it is generally recognized that the so-called "prophylactic" resection, carried out for early enlargement causing few symptoms, with the object of preventing, by better drainage, further enlargement of the prostate, has no justification.

Finally, I should like to refer briefly to a few late results in the series under discussion. In the first place I have several patients in whom, on rectal and cystoscopic examination, the prostate appears to be exactly as it was at the patient's discharge from hospital from two to four years ago.

Secondly, I have some others, rather larger in number, in whom the prostate has definitely increased in size after a completely successful resection.

In one of these the resection was carried out for sudden complete retention. He returned three years later with slight hæmaturia, and otherwise symptomless, but with a prostate so big that a resection would be a most formidable procedure.

Another is more striking, as the degree of enlargement is not merely a clinical impression, but can be expressed in figures. His complaint was of difficulty, culminating in retention of urine, and his prostate was quite small. I removed a small subcervical lobe weighing less than two grammes, with a completely satisfactory result. He returned three and a half years later with difficulty and hæmaturia, and I again removed transurethrally a middle lobe which this time weighed 22 grammes.

Thirdly, I have several patients in whom, though prolonged pre-operative drainage brought about no change at all in the size of the prostate, a large adenomatous gland diminished tremendously in size immediately after an extensive resection, and in whom on rectal examination no enlargement can now be detected, the prostate feeling small and atrophic.

My present belief is that where there is no diminution in size of the prostate with pre-operative drainage, no subsequent shrinkage of the prostate can be expected to occur after a transurethral resection which "canalizes" the prostatic urethra, nor can such a resection be expected to be in the least degree effective in preventing subsequent prostatic enlargement which may again require surgical attention.¹ After a resection of this nature the patient's prospects of immunity from future trouble depend on his expectancy of life and on the rate of enlargement of the prostate. Such a resection would be quite adequate for a man of seventy years of age with advanced cardiovascular disease, and quite inadequate for a man of sixty years of age in good health. On the other hand, following a complete resection in which all the adenomatous tissue is removed, a markedly enlarged prostate will

¹ Since this paper was written a patient in whom a grade I prostatic enlargement was successfully treated by resection three years ago, has again developed complete urinary retention, and has had a grade II prostate removed suprapubically.

become of normal dimensions on rectal examination. Here the prospects of future trouble should be comparable with those after suprapubic prostatectomy, so that a complete resection which with increasing experience becomes more and more often possible, is a sound and logical means of treating simple prostatic enlargement, whatever be the age and general condition of the patient.


SUMMARY.

1. Transurethral resection is indicated, and open operation contra-indicated, in the small fibrous prostate and prostatic carcinoma.

2. It is the treatment of election of the small adenomatous prostate requiring operation.

3. It is preferable to prostatectomy in the aged patient and the poor surgical risk, and to cystostomy where prostatectomy is out of the question. Its indications otherwise are the same as for prostatectomy, the extent of its use depending on the attitude and technical qualifications of the surgeon.

4. When resection is employed in the treatment of simple prostatic hypertrophy, the prospects of permanent cure depend on the completeness of the resection and the patient's expectancy of life.



A NOTE ON THE VARIATIONS OF THE FORAMEN OVALE.

By SYDNEY SUNDERLAND,

Professor of Anatomy Elect, University of Melbourne; late

Honorary Assistant Neurologist, Alfred Hospital, Melbourne.

At the present time many, probably the majority, consider that alcohol injection of the Gasserian ganglion, for the relief of trigeminal neuralgia of the idiopathic type, is a necessary preliminary to surgical division of the posterior root of the nerve. If the pain complained of involves all three divisions of the nerve the ganglion is the objective, but if the condition is limited to the third, or mandibular division, the injection of the nerve in the *foramen ovale* is advocated. The action of alcohol is to destroy any nerve fibres or ganglia with which it comes in contact, and this action is a local one. Thus the fluid has little power of diffusion unless injected in very large quantities, so that, if it is injected some distance from the nerve, there is little hope of its reaching and involving that structure. The injected fluid, then, will only cause interruption of the nerve pathway provided that the needle actually penetrates the nerve or ganglion so as to allow the fluid to pass into and around the individual nerve units, whether these be fibres or cells. Nothing is to be gained by injecting at random in the hope of involving the nerve trunk. Such action is productive of more harm than good, for it will result only in a diffuse fibrositis of the surrounding tissues that will certainly make any future attempts more difficult. Thus to ensure a satisfactory result the ganglion or nerve, as the case may be, must be entered, and this can only be achieved with certainty provided that the *foramen ovale* is located first.

Now it is fully appreciated by those interested in this form of treatment that one cause of failure to produce relief by injection is due either to failure to locate the foramen itself, or to an inability to enter it even when it is found. It is not every foramen that is accessible to the exploring needle. Abnormalities of the *foramen ovale* are fully appreciated, though it is not generally realized that these variations conform to a definite arrangement. The purpose of this short communication is to draw attention to the explanations offered for the presence of such irregularities, and to stress the practical significance of some which have apparently been overlooked.

The phylogenetic story of the *foramen ovale* is an interesting and illuminating prelude to a study of its variations found in adult man. This foramen is developmentally and morphologically a derivative of the *foramen lacerum medium*, which is found at the base of the skull between the petrous temporal and alisphenoid bones. In *Tarsius spectrum* the most primitive condition is present, for the *foramen ovale* is absent and the mandibular division of the fifth cranial nerve leaves the cranial cavity through the general hiatus of the *foramen lacerum medium*. At the other extreme we have,

in the lemurs, a condition in which the *foramen ovale* is situated well forward in the alisphenoid, being separated from the *foramen lacerum medium* by a bar of bone of considerable width. Midway between these two extremes is the transitional type of the non-human primates, in which the foramen is incomplete posteriorly, the bony margin being completed in that situation by the petrous temporal bone. Thus in the monkeys and the anthropoid apes, other than the gorilla, the foramen is present as a notch in the posterior margin of the alisphenoid, which may be widely open or almost enclosed by two spurs of bone passing in from the alisphenoid margin of the foramen. The gorilla presents an unusual type of foramen, for this is now situated along the



FIGURE I. Specimen illustrating incomplete separation of the *foramen ovale* from the petro-alisphenoid fissure.



FIGURE II. Specimen illustrating incomplete separation of the *foramen spinosum* from the petro-alisphenoid fissure, which is also in communication, by way of a suture, with the *foramen ovale*.

suture line between the alisphenoid and squamous temporal bones. The suggestion arising from these observations is that in phylogeny the *foramen ovale* has migrated in from the *foramen lacerum medium* to involve the alisphenoid, and so ultimately to become divorced from its parent foramen.

In the fetus the mandibular nerve passes through the cartilaginous anlage of the *foramen lacerum medium*, thus mimicking the condition in *Tarsius*. With development, ossification occurs along the alisphenoid margin except in the vicinity of the nerve, which appears, therefore, to be migrating forwards from its primitive position to involve the bony margin, which it gradually notches. In this way is reproduced the condition found in the non-human primates (see Figure I). As ossification proceeds, the notch becomes closed around the nerve, so that the next stage is one in which the nerve passes through a distinct foramen in the alisphenoid, though this is still connected to the *foramen lacerum medium* by a suture line which apparently marks the track of passage of the nerve (see Figure II). The

last, and completed stage in man, is that in which the foramen is separated first by a thin bony bar from the petro-alisphenoid line, and, later still, when it is situated well forwards in the alisphenoid, and normally this is not accomplished until after birth. No case has been recorded in which the nerve has passed forwards between the alisphenoid and squamous temporal bones to form the gorilloid type of foramen. Development may be arrested at any one of these stages and thereby give rise to a characteristic and easily explained type of variation. None of these variations, which it should be remembered are common, should, however, give rise to any difficulty in locating the foramen or in entering it once it has been found.



FIGURE III. Specimen illustrating the foramen spinosum in communication with the foramen ovale.

second, that in which the foramen spinosum is incompletely separated from the foramen ovale (see Figure III). In either of these cases damage to the artery becomes a definite possibility when "feeling for", and injecting through, the foramen, and may prevent a continuation of the operation. Despite the closed nature of the infratemporal region, bleeding from this vessel can be considerable. Damage to it is always a possibility, but such an accident is rendered more likely under the circumstances mentioned. The remaining structures associated with the nerve as it passes through the foramen ovale are veins. There is nearly always a venous channel or plexus passing through the foramen to join the pterygoid plexus of veins. The main mass of these veins

In a similar way the middle meningeal artery alters its relation to the foramen lacerum medium, through which it originally passed to enter the cranial cavity, by forming its own bony foramen lateral to the foramen ovale (see Figure II). Of the variations met with in man, the two commonest are, first, that in which the artery passes through the foramen ovale, the foramen spinosum being absent, and,



FIGURE IV. Infero-lateral view of a medial pterygo-spinous plate. The accessory foramina in the plate are for the passage of branches of the mandibular nerve.

may pass through the *foramen ovale*, while on the other hand the greater portion may be responsible for the formation of a new foramen medial to the *foramen ovale*, which is known as the foramen of Vesalius. Damage to these veins probably occurs in every case in which the *foramen ovale* is traversed, although hæmorrhage from such a source is not severe because of the low venous pressure and the density and unyielding nature of the tissues in the infratemporal region.

The most important structure, however, that will influence the passage of a needle through the foramen is a pterygospinous bar. This is a process of bone joining the external pterygoid plate to the *spina angularis sphenoidæ*. In lemurs the bar passes medial to the *foramen ovale*. In the New World monkeys its posterior margin falls short of, or passes medial to, the foramen.



FIGURE V. Specimen illustrating a pterygospinous bar passing lateral to the *foramen ovale*.

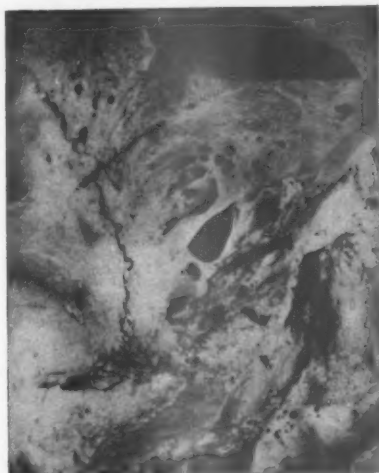


FIGURE VI. Specimen illustrating a pterygospinous bar passing lateral to the *foramen ovale*.

The bar passes lateral to the foramen in the Old World monkeys, and is pierced by branches of the mandibular nerve as they pass out from the main trunk which continues down deep to the bar. A pterygospinous bar is by no means uncommon in man, and when present it usually passes medial to the foramen, thus repeating the lemurine and platyrrhine conditions (see Figure IV). It may be complete and extend to reach the spine of the sphenoid, or incomplete, in which case it fades away at the base of the skull before reaching that bony process. A bar so placed forms no barrier to the entry of the foramen unless its formation so rotates the plane of the foramen that it can no longer be approached and entered along any of the recognized routes. Usually, however, when the bar is reproduced medially the plane of the foramen is so rotated that the orifice comes to face directly laterally or infero-laterally, and this renders it particularly accessible. Now the position of the foramen, no matter what its situation, can only be appreciated

if the bony margin is located first. Under such circumstances the foramen can be so easily reached that, unless great care is exercised, the entry passes unrecognized, and if the needle is pushed onwards, in the hope of locating a



FIGURE VII. Specimen illustrating a complex pterygospinous bar. The bar divides into two processes which pass back to straddle the foramen ovale.

this arrangement is not commonly met with in man (see Figures V, VI and VII). When complete and well developed it forms an efficient obstacle to injection. Even if it is incomplete and fades away on the lateral side of the foramen, it may so rotate the plane of the foramen that that orifice becomes directed inwards away from the path of an exploring needle. The bar may be present as a plate or as a low crest of bone along the base of the skull, and in the former case the obstruction will always be greater than in the latter, in which a very oblique infero-lateral approach may locate the foramen by the needle passing under the crest. The base of the bar or plate is always perforated opposite the foramen ovale to allow the passage of branches of the mandibular nerve laterally,

foramen that has already been traversed, then the cavernous sinus or internal carotid artery will be punctured or the posterior cranial fossa entered beneath the attachment of the *tentorium cerebelli*. Such an error is serious enough, but if alcohol is now injected with the needle in any of these positions much damage will result. Harris makes it a rule never to continue through the foramen for more than 0.6 centimetre (a quarter of an inch), but such judgement can only be exercised provided that the margin of the foramen is carefully outlined first. These readily accessible foramina, whose accessibility is brought about by an alteration of the plane of the bony basis of the foramen, are also found in the absence of any pterygospinous bar, and their occurrence is an indication to observe caution in all cases.

The Simian condition may, however, be reproduced and the bar be directed lateral to the foramen, though

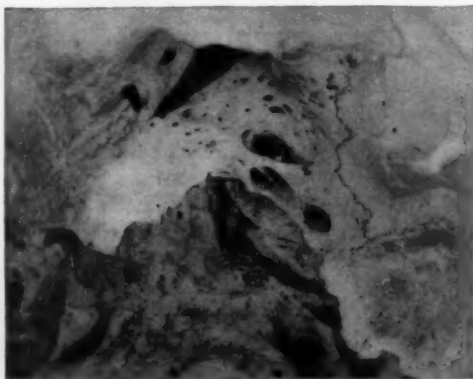


FIGURE VIII. Infero-lateral view of the condition seen in Figure VII to illustrate the accessibility of the foramen ovale.

the main trunk continuing on deep to the plate. In Figure VIII a single foramen is illustrated beneath a fine spur of bone, and here this foramen is so large and the bar so thin that the *foramen ovale* is in full view from lateral and infero-lateral aspects and could be readily entered above the bar along any of the recognized approaches. It is instructive, on the other hand, to compare this condition with that illustrated in Figure IX, which is an infero-lateral view of the arrangement seen in Figure V. It will be readily observed that, in this case, there is no chance whatsoever of entering the *foramen ovale* from the lateral or infero-lateral aspect. It is these cases that present the greatest difficulties and in which no degree of perseverance on the part of the operator will result in success.

The osseous bar may be replaced by a pterygospinous ligament, which is a very tough structure, and this, if passing lateral to the foramen, may render the approach to it a very difficult procedure indeed. Two aberrant muscle

slips are occasionally found in this region. One, the pterygospinous muscle, stretches between the posterior margin of the external pterygoid plate and the spine of the sphenoid or tympanic plate of the temporal bone, while the other, the *pterygoideus proprius*, passes from the infratemporal crest of the alisphenoid, over the external pterygoid muscle to its insertion on the posterior margin of the external pterygoid plate. The latter muscle is always lateral to the *foramen ovale*, whilst the former is usually medial, though it may pass laterally to it. These muscles may contain a large amount of tendinous material in their substance, which will considerably hinder an approach to the foramen, particularly if an indiscriminate injection of alcohol on a previous occasion has resulted in a surrounding fibrosis with increased thickening of the structures enumerated.

From an examination of this series of variations it becomes apparent that entrance to the foramen is by mere chance alone. Nevertheless, the so-called normal arrangement is so common, and the foramen so accessible, that trouble is not often encountered, though caution is always demanded in every case. The variations outlined, however, do account for those occasions when, even in skilled hands, all attempts to reach and penetrate the foramen are defied.

ACKNOWLEDGEMENT.

I wish to thank Mr. H. Marriott for preparing the photographs which accompany this paper.



FIGURE IX. Infero-lateral view of the condition seen in Figure V, to illustrate the inaccessibility of the *foramen ovale*.

EARLY INDICATIONS FOR SURGERY IN HEAD INJURIES.

By I. DOUGLAS MILLER,
Sydney.

THE classification of head injuries into those affecting the skull and those affecting the brain is not an altogether artificial distinction, for it is well known that the brain may be even fatally damaged without injury to the skull, although, on the other hand, it is more than likely that the presence of a fracture will imply violence of a degree likely to injure the brain.

There is no doubt as to which group should absorb our most careful attention, and it is now over a century since John Bell thus emphasized this point:

We have this encouragement to consider the pathology in place of the anatomy of the skull as a rule of our prognostics, that while anatomy and the enumeration or classification of the fractures have led to an undue propensity to operation, the study of the living powers, and the mutual dependence of these parts, leads to a reserved, modest and rational practice, to a just confidence in the power of nature, to a careful and solicitous attention to all the insidious symptoms of an injured brain.

It is interesting to reflect that so long after such sound and authoritative advice was first given, our hospitals and courts often tend to be more concerned over the anatomical than the pathological aspects of head injuries. The surgical treatment of skull fractures will therefore be briefly discussed, and more attention given to other intracranial injuries.

DEPRESSED FRACTURES.

The great majority of depressed fractures are compound, and therefore call for the treatment accorded to any compound fracture. There is no doubt that in dealing with these conditions it is the wound and not the depressed bone that matters, and the essential feature of treatment is the scrupulous *débridement* of damaged scalp, skull, dura and brain.

Often there arises the difficulty of deciding whether some scalp wound is in fact part of a compound fracture. In these cases it is best to infiltrate the scalp with a little local anæsthetic solution and open the wound more widely for purposes of exploration. Where the fracture is of the punctured variety and the bone is obviously driven in and soiled, there is no doubt that it should be removed or elevated. In those cases, however, where the depression is pond-shaped and the bone clean, interference with the bone is by no means imperative, and if thought desirable, in view of the possibility of introducing infection, attempts at elevation may well be omitted. In those rare instances where a depression of bone exists without injury to the scalp, we may well consider whether operation is indicated at all.

One may say that a depression of the skull will only cause focal symptoms in those rare instances where the depressed area is actually overlying a highly important functional centre. In most cases it causes no symptoms, and the only indication for its elevation is to obviate the possibility of late

sequelæ or for cosmetic reasons. There is certainly no urgent indication for the elevation of such a fracture, and there is but little reason for the fear that epilepsy may be a late sequela. The type of injury which is apt to result later in epilepsy is rather the injury which, having involved a local area of dura and cerebral cortex, later results in an adherent meningo-cerebral scar.

In these cases of depressed fracture it is rather remarkable that the great majority of patients do not appear to suffer much general cerebral damage. Very few have a long period of unconsciousness, and many are conscious on admission to hospital.

INTRACRANIAL CONDITIONS REQUIRING EARLY SURGICAL INTERVENTION.

The intracranial conditions requiring early surgical intervention are the various types of epidural and intradural hæmorrhage, the hydromas, and occasionally the generalized œdema which is resistant to other forms of treatment.

In an analysis of a large number of head injuries we find that those wherein surgery may be indicated or debated are indeed small in number. For convenience in discussion of this class of case I have chosen depth of unconsciousness as a basis for classification.

First, there is the class of patient who on admission to hospital is deeply unconscious, with cyanosis and stertorous breathing. It is common experience that the majority of such patients die within a few hours. Out of twenty-five patients admitted to St. Vincent's Hospital, Sydney, and classified as deeply unconscious, twenty-two died.

This group forms a small proportion of all head injuries. My twenty-five cases are selected from a series of 718 patients admitted in an unconscious condition or with a history of unconsciousness. In this series there was a mortality of thirty-five, and twenty-two of these died in less than twenty-four hours after admission to hospital. All these, as stated above, were deeply unconscious on admission and remained unconscious. The remaining thirteen survived for periods ranging from two to fifteen days, and during this time either remained unconscious or passed through phases of partial recovery followed by relapses into unconsciousness.

In regard to the first type of case, *i.e.*, those admitted deeply unconscious and dying within a few hours, autopsy reveals gross laceration of the brain, of such a nature that no form of surgical procedure could alter the ultimate issue. Surgeons have passed through a period in which they essayed to save such patients by opening the skull in the hope of producing a decompressive effect. This is always futile, as the underlying cause of unconsciousness in these cases is not in the nature of a compression.

Having excluded this class of case, which forms the greatest mortality group, we are left with 683 patients who went on to recovery and thirteen who died. In the 683 cases there were three operated on for clot compression and a small number had late decompressive operations. The vast majority went on smoothly to recovery, and at no time were considered as likely to require surgery.

The question presents itself whether any of those who died in the late mortality group might have been saved by surgical measures.

Unfortunately, owing to the workings of our coroner's department, the answer to this question usually remains one of conjecture, and we can only

draw conclusions from *post mortem* figures collected elsewhere. From a clinical point of view these patients run a course during which they may be unconscious all the time, or may have a period of partial recovery and then become unconscious again.

There are two pathological conditions which may bring about this picture. The first is damage to vital centres which, though not immediately fatal, may prevent the long continuance of existence. Such patients may live for days or weeks, and then gradually die, though there is no evidence of gross intracranial damage or hæmorrhage. The other condition is clot compression and occasionally compression by pathological collections of subdural fluid. It is these latter conditions which are occasionally overlooked and comprise the preventible mortality group.

That epidural and subdural hæmatomata are overlooked is amply testified to by the facts of several *post mortem* series. In 100 *post mortem* examinations Jefferson found nine unrecognized subdural hæmatomata. In a similar series of 512, Vance found 132 subdural hæmatomata, and McCreedy and Berry, in 520 cases of fractured skull examined *post mortem*, found 51 unrecognized subdural hæmatomata. We may take these figures, therefore, as evidence that in any series of cases, epidural and subdural hæmatomata are apt to be overlooked.

Epidural Hæmatoma.

Every medical student is traditionally instructed in the recognition of a classical epidural hæmatoma. This emphasis of a complete picture both exaggerates the frequency of the complaint and misleads in the recognition of any hæmorrhage that does not follow the classical train of events. True, indeed, one may see the sequence of loss of consciousness, lucid interval, and then further loss of consciousness, all classically accompanied first by heterolateral and later by homolateral irritative and then paralytic phenomena, but it is rarely that the clinician or any other trained observer is standing by to make such observations.

In my own small series I have always been called to see an already deeply unconscious patient. The history that the patient gradually became unconscious after a head injury, is almost sufficient indication of hæmorrhage, and signs of external violence such as a temporal hæmatoma, or some evidence of weakness on one side, are adequate indication of the side of the hæmorrhage. The presence of a temporal fracture is evidence in favour of an epidural hæmatoma. In such cases where the presence of hæmatoma is suspected, an exploratory bur hole is a simple and safe procedure, and the indications for it are in fact urgent.

In Jefferson's series of *post mortem* examinations following death from middle meningeal hæmorrhage, the survival period varied from sixty-five minutes to three days. It is obvious that patients in the former category could not have been afforded surgical relief, but his findings serve to point the moral that the sequence of events leading to death in epidural hæmorrhage may be so rapid as not to be recognized.

In passing, it may be well remarked that just as the frequency and ease of diagnosis of middle meningeal hæmorrhage are grossly exaggerated, so, too, is the treatment apt to be imagined as too simple. It is an easy matter to trephine and tie the middle meningeal artery, but it may be a very difficult matter to deal with a large epidural hæmatoma. As the hæmatoma increases

in size, it strips a wide area of dura from the bone, and tears other small vessels, which in their turn continue to ooze. It is essential to control all these points, and it has seemed to me always desirable to have the rather wider access afforded by a small flap, and to have at one's disposal all the devices used to control hæmorrhage in special intracranial surgery. In view of constant warnings, the epidural hæmatoma is less likely to be missed than the subdural variety.

Subdural Hæmatoma.

All are familiar with the chronic subdural hæmatoma which may follow upon a head injury and give rise to vague symptoms of cerebral damage within days, weeks or months, but all are not equally aware of that acute type of subdural hæmatoma, the early recognition of which may save life. In his series of *post mortem* examinations, Jefferson found that the period of survival in these cases varied from seventy minutes to sixteen days. Five of the nine patients lived for days, and in these at least recognition of the underlying pathology might have saved their lives. It should be emphasized that just as we no longer think of operating in the hope of saving the grossly lacerated brain, so we must think much more of operating on that class of patient who may prove to have a hæmatoma.

Most of these patients are admitted in a semiconscious condition and become unconscious, or, having been admitted unconscious, pass into a deep coma. We have, in our year's series, twelve patients who have run this course, dying without operation and not having had *post mortem* examinations carried out. The cause of their deaths is a mystery, and may well have been subdural hæmatoma.

Bearing in mind, then, the fact that some of the patients who die might always have been saved if hæmatomata were recognized, we have to consider the criteria which should lead us to a decision to explore the skull.

State of Consciousness.—As already mentioned, almost all patients admitted deeply unconscious die rapidly and nothing can save them.

There are next the patients admitted dazed or stuporose or even conscious, who ultimately become deeply unconscious and die. There is no doubt that in the observations of such patients the clinical sign of paramount importance is a change in the depth of unconsciousness. It is desirable to have some standard of assessing this depth, such as being able to rouse the patient by speaking, shouting, and various grades of painful stimuli. Every nurse in charge of patients suffering from head injuries should be able to assess and recognize a deepening of the unconsciousness.

Power and Sensation.—Though as a rule an intracranial hæmorrhage will cause coma and marked loss of power, it is a common occurrence to find the loss of power preceding the coma and giving a very reliable lead to localization. When a stuporose patient is first seen, observations should be made of involuntary movements which may and must, if necessary, be initiated by painful stimuli. A stuporose or lightly comatose patient will always attempt to remove an annoying pin point from his lips. In this way, first one limb and then the other should be tried. Hours later, when the patient's condition may have changed, one may obtain valuable information from repeating this test. This examination may also indicate that sensation of pain does not appear to be appreciated in certain regions.

Aphasia.—In this class we must consider the patient who will not answer questions, though he appears to hear and understand. This sign, often misinterpreted at first as stupidity or hysteria, may be of the utmost localizing value.

Eye Grounds and Fields of Vision.—It is a well recognized fact that papillœdema may develop very rapidly as the result of a sudden increase of intracranial tension. This may occur in a period of hours, and should be looked for, as it may give information not only as to intracranial tension, but also as to lateralization. The fields of vision can often be estimated roughly, even in the stuporose and uncooperative patient, by noting that he is inattentive on one or other side. This likewise may be of localizing value.

Pulse Rate.—In the observation of patients with head injuries, it is customary to give the pulse record great importance, even to the point of almost exclusive attention. It does, however, appear to be a fact that it is difficult to draw any conclusions from the pulse rate.

It is generally assumed that a falling pulse rate indicates a rising intracranial pressure. There are, however, many other factors, such as shock and recovery to the normal pulse rate, and the variations of normal, which tend to confuse the value of these findings. Actually, on analysing the half-hourly pulse charts of a large series of cases, I am unable to draw any conclusions from them. The charts have shown that patients dying at different periods have widely varying pulse rates. It might be said that the chief value of the half-hourly pulse chart is as an assurance of the patient's regular supervision. If at the half-hourly visits the nurse also made observations on the state of consciousness and presence of movements, her chart would be more valuable.

Blood Pressure Readings.—Blood pressure readings have often been advocated in the periodical observation of head injuries. This method of observation would appear to be of even less value and more fallacious than any pulse observations, there being associated with a head injury so many factors capable of disturbing the blood pressure, which, added to the lack of knowledge of the normal blood pressure for the individual, would make these observations of little value and possibly misleading.

Lumbar Puncture.—There are few, if any, indications for lumbar puncture within the first twenty-four hours. It has a decided danger in the presence of intracranial bleeding. We have seen that the immediate mortality within twenty-four hours is due to gross injury and occasionally to massive hæmorrhage. Lumbar puncture can only influence these conditions adversely. In other cases it can be used only to estimate the pressure of the cerebro-spinal fluid, or betray the presence of blood, and as most individuals would be resistant to the manœuvre, in their usual irritable condition, its manometric value would be negated. In difficult cases where compression is suspected, lumbar puncture may give some information, provided that it is done with a manometer.


On the one hand, a quite massive hæmatoma may give no indication of its presence at lumbar puncture, and, on the other hand, blood in the cerebro-spinal fluid may not indicate a hæmatoma. The presence of blood is therefore not evidence one way or the other of intracranial hæmorrhage.

I therefore feel that as a diagnostic aid lumbar puncture is of the most limited value and is fraught with grave danger.

Radiological Examination.—As previously remarked, radiological examination of the skull may be helpful in the diagnosis of middle meningeal hæmorrhage, as this condition is so frequently associated with a fracture.

Exploratory Bur Holes.—In cases where there is reason to suspect a hæmatoma, an exploratory bur hole will be an easy, safe and certain procedure. Any extradural hæmorrhage will be disclosed by a bur hole in the temporo-parietal region. If no extradural hæmatoma is found, the dura must be opened, and if there is a subdural hæmatoma on that side, it will almost certainly have extended this far forward. If the findings on one side are negative, the procedure may rapidly be repeated on the other.

There can be no doubt that this is the correct procedure where intracranial hæmatomata are suspected. With a simple technique it is easily and quickly performed, and the surgeon knows immediately whether he is dealing with a condition that he can cure. If the findings are negative, no risk has been run, and one can feel content in having excluded the curable fatal lesion.



PNEUMOCOCCAL PERITONITIS.

By A. EISDELL MOORE,
Auckland.

THE object of this short paper is to bring to the notice of the Fellows of the College the extreme value that is apparently to be obtained in pneumococcal peritonitis by the administration of Felton's antipneumococcal serum.

Pneumococcal peritonitis is not a common disease, and some indication of its relative infrequency can be obtained from the literature and from the reports of various speakers at surgical congresses at which the subject of pneumococcal peritonitis has been discussed. Horne⁽¹⁾ recorded seven cases, stating that 2% of abdominal emergencies in childhood were due to this disease. At the meeting of the New York Academy of Medicine in April, 1934, Dr. Murray H. Bass⁽²⁾ reviewed the nineteen cases that had been seen in twelve years at the Mount Sinai Hospital. At the British Medical Association meeting in 1932, Barrington-Ward⁽³⁾ reviewed twenty cases that had occurred in Great Ormond Street Hospital in the previous fifteen years, and quoted Duncan's thirty-four cases in the Children's Hospital in Toronto over a period of ten years. At the same meeting Spence⁽⁴⁾ referred to twenty-one cases at Newcastle in five years. Thus one would conclude that an average of only three or four cases would be admitted to any large hospital in a year. During 1936 there were five cases treated in the Auckland Hospital.

With regard to the question of mortality, all authorities agree that the rate is very high, the figures varying in the articles referred to from 40% to 90%. Choyce's "Text-Book of Surgery" places the mortality at 75%, Nelson's "Loose-Leaf Surgery" in the neighbourhood of 60%. Bass, referring to his cases at Mount Sinai, uses these words: "The mortality in this series is the same as that found in several hundred previously reported, that is, nearly 58%."

Of the five cases admitted to the Auckland Hospital in 1936, four were admitted to the surgical side, and after operation were treated with large doses of serum. All these four patients survived, and although one realizes that a series of four consecutive successful cases is not sufficient to substantiate any extravagant claims for serum, yet in a disease with a mortality of about 60%, the treatment adopted to produce such a result is worthy of investigation. The fifth patient was admitted to the medical side, and not referred to a surgeon till the twenty-fifth day, when a large abscess was drained. The patient developed acute intestinal obstruction ten days later and died.

There is no need on this occasion to discuss the disease in general, but a few points may be mentioned, especially regarding the method of infection.

The pneumococcus may reach the peritoneum, first, and rarely, by extension from the pleura; secondly, and occasionally, by means of the blood stream, from infection in joint or lung, and, thirdly, and most commonly, by direct extension from the Fallopian tubes.

Pneumococcal peritonitis is essentially a disease of childhood, affecting the female far more frequently than the male, with a common age incidence of about five or six. The Auckland cases recorded are all in little girls aged from four to twelve years, with an average age of seven. The facts that patency of the vagina may not be complete till the third year, and that after the seventh or eighth year the vaginal secretion becomes acid and unfavourable to the pneumococcus, help to explain the age incidence.

The symptoms are those of a primary pelvic peritonitis, and naturally appendicitis has always to be considered in the differential diagnosis. The following points, however, are of importance:

1. Absence of a typical history of appendicitis.
2. Tendency to diarrhoea.
3. Abnormally high temperature.
4. Unusual abdominal distension, with widespread tenderness.
5. Raised respiration rate, which is of frequent occurrence.
6. Abnormally high leucocytosis, which is occasionally present.

It can, however, safely be said that no clinical symptom is of sufficient value to enable one to arrive at a trustworthy diagnosis. Non-intervention is too risky, and confirmation of the diagnosis by a limited right iliac exploration is the safest course. Abdominal puncture with aspiration of a few drops of exudate for examination has been strongly advocated, but would seem to have little advantage over a very limited exploration under local anaesthesia. The pathological changes found at operation are fairly characteristic, and are often the first intimation to the surgeon that the case is one of pneumococcal peritonitis. The exudate is profuse, odourless and greenish-brown in colour, and contains many flakes of fibrin. The peritoneum is red and thickened and the coils of intestine are often adherent to one another, with large flakes of fibrin.

When the diagnosis has been made at operation, different operative procedures have been carried out in the various series of cases reviewed, but the mortality does not seem to have been influenced by any special procedure adopted. In the four cases operated upon early in this series, two had appendicectomy and pelvic drainage, one appendicectomy and closure, and one simple pelvic drainage. What, however, one does feel has influenced the mortality has been the administration of large doses of Felton's serum post-operatively.

The case histories of our Auckland cases are as follows:

CASE I.—S.A., aged five years, was admitted to hospital on February 12, 1936, with a history of abdominal pain and vomiting for nine hours. The temperature and pulse rate were high, the abdomen was rigid and exquisitely tender, and the child was delirious. The presence of pneumococcal peritonitis was suspected, and exploration was carried out under local anaesthesia. Free pus of typical nature was found, from which pneumococcus, type I, was obtained by culture later. The pelvis was drained and 20,000 units of Felton's serum were given through the drainage tube, a further 60,000 units being given intravenously during the subsequent three days. The patient's improvement was dramatic. The temperature and pulse rate were almost normal on the second day, and the child was wanting to stand up in her cot on the third. No complications occurred, and the patient was discharged in twenty-three days.

CASE II.—P.B., aged twelve years, was admitted to hospital on March 14, 1936, with a history of abdominal pain, vomiting and diarrhoea. The temperature and

pulse rate were high, and the abdomen was rigid and tender. Operation was performed, and findings typical of pneumococcal peritonitis were obtained. Free pus was present, from which pneumococci were subsequently obtained by culture. Appendicectomy and pelvic drainage were carried out. Thirty cubic centimetres of Commonwealth serum were given intramuscularly, and 130,000 units intravenously and 10,000 intraperitoneally during the next five days. The patient was seriously ill for four days, but gradually improved, developed no complications, and was discharged on the thirty-eighth day.

CASE III.—H.D., aged six years, was admitted to hospital on April 13, 1936, with a three-day history of abdominal pain and vomiting. The temperature and pulse rate were high, and great abdominal tenderness was present. Operation the same day revealed typical pus, from which pneumococci were obtained on culture. Appendicectomy was performed and the pelvis drained. Ninety thousand units of Felton's serum were given intramuscularly and 80,000 intravenously in seven days. This patient ran a hectic temperature for two weeks and the wound discharged profusely. The earlier injections were given by the intramuscular route. One felt that the intramuscular injections were far less beneficial than the intravenous, as improvement began when the latter route was adopted. A large abscess formed in spite of the pelvic drain, and this had to be evacuated on the sixteenth day. From then improvement took place, but acute intestinal obstruction developed on the thirty-eighth day. Operation for this was performed under spinal anaesthesia, with recovery, the patient leaving hospital in good health on the sixty-seventh day.

CASE IV.—C.P., aged six years, was admitted to hospital on July 11, 1936, with a twenty-four-hour history of abdominal pain, vomiting and diarrhoea. The temperature and pulse rate were high, and the abdomen was tender and rigid. A diagnosis of acute appendicitis with perforation was made, and operation was performed immediately. Typical findings of pneumococcal peritonitis were present, and the organism was obtained on culture. Only 10,000 units of Felton's serum were given to this child in the first twenty-four hours after operation, and at the end of that time her condition was desperate. She had a temperature of 40° C. (104° F.) and a pulse rate of 168, and was blue and almost moribund. Administration of serum was then pushed, 10,000 units being given every four hours, and by the next morning the temperature was normal and the pulse rate 132. Gradual improvement continued, and the child was out of immediate danger within a week. A residual abscess, causing some subacute intestinal obstruction, required drainage on the fourteenth day, but the patient left hospital in good health on the thirty-fifth day.

It is obviously quite incorrect to draw conclusions from so limited a series of cases, but one feels that in a disease with an accepted mortality of about 60%, four consecutive cases without a death is an indication to study any special treatment given, and one feels that the big doses of Felton's serum were life-saving in two at least of these cases.

In conclusion, one would say that if pneumococcal peritonitis is the provisional diagnosis, non-intervention is too risky, and that limited exploration under a local anaesthetic is the correct treatment. If the appearances seen at operation are typical, then a swabbing should be taken for examination, the excess of exudate cleared from the peritoneal cavity by suction and pelvic drainage instituted. Felton's serum, in dosage of 10,000 units intravenously, should be given every four hours till the critical stage is over, which will probably have occurred within twenty-four hours. Further doses should be given at longer intervals during the next three days.

When the stage of acute toxæmia is over, the patient has to be watched for localized collections of pus, which may burst into an old drainage track or require exploration, while throughout convalescence there is a continuous risk of intestinal obstruction from coils of bowel becoming adherent.

ADDENDUM.

The above paper was presented at the meeting of the Royal Australasian College of Surgeons at Auckland in January, 1937. A further six cases can now be added to this series. Four of these were admitted to the Auckland Hospital, and two were seen in private practice.

I wish to express my gratitude to Mr. Furkert for searching the hospital records, and for providing the reports on the hospital cases; and also to Mr. Smith Gray for permission to include in this series the cases admitted under his care.

The four patients admitted to the Auckland Hospital from January, 1937, to August, 1938, were all little girls of ages four, five, five and seven respectively. Three were treated by drainage and serum, and one by drainage, serum and appendicectomy. There were three recoveries and one death. In the fatal case the serum was administered in one massive dose, and was followed by a mild anaphylactic reaction. Death occurred within about six hours, apparently from severe toxæmia. From all cases a pure culture of pneumococcus, type I, was obtained.

Of the two private cases, one, referred by Dr. E. H. Bryant, was dreadfully ill when first seen: cyanotic, incontinent, with a temperature of 40.3°C . (104.5°F .) and a pulse rate of 130; and the other was seen in consultation with Dr. Graham Lindsay after he had done an emergency operation for acute peritonitis and recognized the typical pneumococcal appearances. In both cases pneumococci were obtained on culture, both patients were given big doses of Felton's serum, and both, after a few days of grave anxiety, made complete recoveries.

Mr. Furkert has searched the records of the Auckland Hospital in the years 1930 to 1936, and gives the following table:

	Cases.	Deaths.	Mortality.
Primary pneumococcal peritonitis treated without adequate Felton's serum	11	11	100%
Late localized abscess cases	4	1	25%
Total	15	12	80%

From January, 1936, to August, 1938, the following table can be compiled:

	Cases.	Deaths.	Mortality.
Primary pneumococcal peritonitis cases in Auckland Hospital treated with adequate Felton's serum	8	1	12½%
Cases in private practice in 1938	2	0	—
Total	10	1	10%

One can therefore record a series of ten cases of acute pneumococcal peritonitis with only one death, a mortality of 10%, which is, I believe, far lower than that in any other series of cases yet published.

It is remarkable that all the cases in which the pneumococcus has been typed (eight of the series) have shown pneumococcus, type I, which suggests that this type has a special predilection for the peritoneum, and doubtless this is the reason for the good results obtainable by serum.

It is apparently essential that the serum be given intravenously. In several cases where, because of the mechanical difficulty of administration, intramuscular injections were employed, a clinical relapse was observed, but this was in each case controlled by reverting to the intravenous method.

REFERENCES.

- ⁽¹⁾ Cyrus F. Horne: "Pneumococcus Peritonitis", *Annals of Surgery*, Volume cii, 1935, page 391.
- ⁽²⁾ Murray H. Bass: *The Medical Clinics of North America*, Volume xix, 1934, page 847.
- ⁽³⁾ L. E. Barrington-Ward: "Pneumococcal and Streptococcal Peritonitis", *The British Medical Journal*, October 15, 1932, page 704.
- ⁽⁴⁾ J. C. Spence: British Medical Association meeting, Section of Diseases of Children, *The Lancet*, August 13, 1932, page 343.

Surgical Technique.

THE USE OF A DIATHERMY LOOP IN THE EXCISION OF PILONIDAL SINUSES.

By A. EISDELL MOORE,
Auckland.

PILONIDAL sinuses are by no means uncommon, and during the last few years there have been several articles written regarding the condition. In nearly all these articles stress has been laid on the difficulty of complete eradication of the trouble and the comparative frequency of recurrence.

All authorities agree that the condition is one of developmental defect, in which there is faulty agglutination of the fetal skin folds. There is some variance of opinion, however, as to which particular embryonic layer is actually at fault, but the most commonly accepted view is that if the two folds meeting in the mid-line fail to establish an even epidermal union, then a "pouching in" at this line occurs. This may show, according to its depth, as (a) a groove, (b) a blind sinus or (c) a cyst separated from the overlying skin.



FIGURE I. Situation of sinus.

Clinically, the condition is a skin-lined cyst or sinus, lying between the buttocks and over the sacrum. It is more commonly found in men than in women, and most of the patients are found to be between fifteen and twenty-five years of age when first seen. Advice is almost always sought because of irritation, pain, or the discharge of pus. If a cyst is present, the lump has been there since birth, and the patient has become used to it. If the condition is of the pure sinus type, the orifice of the sinus, which may be very small, may never have been noticed by the patient, even though a tuft of hair may be protruding from it.

On examination, either a cyst or a sinus is found situated in the characteristic position stated above, and almost invariably shows inflammatory changes, with the discharge of particularly offensive pus from the sinus, and reddening of the surrounding skin. Where multiple sinuses are present, or where the main opening is away from the mid-line, the sinuses are lined not with skin, but with granulation tissue, and are due to infection of a pilonidal tract, with multiple "pointings".



FIGURE II. Diagram of sinus.

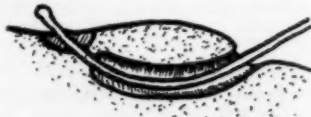


FIGURE III. Probe passed and incision made onto its point.

As the deformity practically invariably comes for surgical examination only in an inflamed state, a differential diagnosis has to be made from furunculosis, tuberculosis, syphilis and even *fistula-in-ano*.

As regards treatment, all authorities are agreed that complete extirpation is essential. Lahey⁽¹⁾ says that all extensions of the sinuses, laterally and subcutaneously, to the fibres of the *gluteus maximus* on either side of the sacrum must be dissected

away cleanly. Cattell and Stoller⁽²⁾ have reported fifty-nine cases and the end-results of forty. Recurrence after operation in their series was very common.

As the condition is associated with infection, extensive clean dissection often leads to a big infected wound with a long period of healing. Furthermore, knife dissection is often hampered by bleeding.



FIGURE IV. Diathermy division of skin.



FIGURE V. Diathermy removal of sinus by wire loop.

In the last three years I have operated upon six cases, utilizing a diathermy loop with cutting current, and each has healed by primary union. The method I have used is as follows.

The skin area is cleansed with ether and iodine, and the sinus is wiped out with a probe dressed with "Acriflavine" in 0.1% solution. A clean probe is now passed into the sinus for its full length, when the end of the probe is invariably felt to be either in a cyst or in the blind end of the sinus, which is dilated as a result of the secondary infection and fairly close under the skin. The end of the probe is cut down upon with the diathermy cutting current, and the probe brought through, so that the whole of the epithelium-lined sinus is in contact with the probe. Then with a cutting diathermy current the overlying skin is divided, but care is taken not to expose the probe. The diathermy loop is now passed over the probe and, with the cutting current switched on, the loop is drawn back till the probe is free, surrounded by a tube of the epithelium-lined sinus. There should be no hole in this sleeve of tissue. In this way, contamination of the wound is avoided and immediate suture can safely be performed.



FIGURE VI. Sleeve of tissue removed.

In addition to the suturing of the skin margin, one or two deep silkworm-gut sutures should be introduced to prevent any tearing open of the wound. With these the wound can be sealed with collodion and the patient treated as ambulatory.

In none of the six patients in whom this technique has been employed has there been any trouble in obtaining primary union.

References.

⁽¹⁾ Frank H. Lahey: "A Further Suggestion for the Operative Treatment of Pilonidal Sinuses", *Surgery, Gynecology and Obstetrics*, Volume liv, March, 1932, page 521.

⁽²⁾ R. B. Cattell and L. W. Stoller: "Treatment of Pilonidal Sinus and End Results", *New England Journal of Medicine*, Volume ccvi, January 21, 1932, page 110.

Lester Breidenbach and H. Lyon Wilson: "Pilonidal Cysts and Sinuses", *Annals of Surgery*, Volume cli, September, 1935, page 455.

Case Reports.

MULTIPLE DUODENAL, INTESTINAL AND OTHER ABNORMALITIES.

By HUGH R. G. POATE,

Sydney.

THE patient, a male, aged twenty-two years, was first seen on January 30, 1936, and complained of attacks of acute abdominal pain with severe vomiting and great prostration at intervals over a period of many years. The attacks had increased in severity and frequency over the past twelve months, during which time he had lost over 6.3 kilograms (one stone) in weight.

The attacks came on without warning, and were unrelated to the taking of food. The patient found that the quickest and easiest way of obtaining relief was to abstain from taking any nourishment or even fluids for twenty-four hours and to go to bed; in fact, the attacks were so violent that it was impossible for him to get about while one was in progress.

His parents stated that ever since birth he had been subject to attacks of vomiting, and that in his early years he had been treated for congenital pyloric stenosis. When he was seven years old he had a laparotomy performed by an eminent surgeon, and I am indebted to Dr. G. H. S. Lightoller for the following notes and the radiological report



FIGURE I. Tracing of skiagram, July, 1921.

of July, 1921, a tracing of one film being reproduced (see Figure I). The report (July 22, 1921) is as follows:

"An opaque meal was administered with the following findings: The stomach is irritable at first, subsequently showing fatigue. There is a large six-hour residue and a small twenty-four-hour residue in the stomach. This residue appears more likely to be due to pylorospasm than to actual pyloric stenosis.

"A congenital abnormality is present in the colon. The caecum is situated in the mid-line near the pylorus, and the colon then passes down into the pelvis and up to the left as shown in the skiagrams. The examination was not carried out beyond thirty hours, but there is evidently some condition about the level of the umbilicus which fixes the caecum and colon and probably the stomach. The presence of such a lesion would account for the patient's gastric and intestinal stasis."

On July 26, 1921, the abdomen was opened by a left paramedian supra-umbilical incision. The stomach and the first part of the duodenum immediately protruded and were lifted out, both being very dilated and the pylorus easily admitting a thumb; the duodenum was 1.25 to 1.87 centimetres (half to three-quarters of an inch) in

diameter. The large bowel was then inspected and was found lying quite free in the abdomen, with a very long mesentery. The whole of the large bowel from the caecum to the sigmoid colon was easily lifted out through the wound; the appendix was long and not inflamed, and the bowel was collapsed. The large bowel was then returned to the abdomen and the small intestine lifted out. This was also collapsed. The mesentery was very free, but not very long, and suggested the presence of a partial torsion as its apex was only about three centimetres (an inch and a quarter) in breadth. The third part of the duodenum was also inspected and found dilated like the first part. A more or less complete blockage was caused at the duodenal-jejunal junction by several membranous bands, which were removed, the intestine then being returned to the abdomen. The caecum was again lifted out and the appendix removed. The distended stomach and duodenum were squeezed, forcing air easily into the small intestine, and the abdomen was then closed. It was noted that the membranous bands mentioned were not adhesions, but of congenital origin. A very noticeable feature was the presence of very large vessels and enlarged mesenteric glands.

As regards the patient's previous history, his father said that the child had remained for as long as four months without an attack, but that usually the intervals were shorter. For several weeks previous to operation he had not been wholly free from discomfort, although there had been no vomiting, and the pain had on occasions been severe. Usually he vomited copiously and then became hungry. The patient's appetite when he was well was usually voracious. He had been subject to attacks of bronchitis, and this factor had to be considered in relation to the duration of anaesthesia when he underwent operation.

Within nine months of the operation the patient is reported to have had two severe seizures, but for the next seven years he was somewhat better, although not free from attacks of abdominal pain. He then developed crises of violent abdominal pain with copious vomiting and great prostration.

Dr. H. M. Hollingworth in January, 1936, stated that in the last two years the attacks were becoming more frequent and apparently more severe, with minor attacks of pain in between the major attacks. The course of a typical major attack was as follows. The patient developed severe pain, with acute onset, in the lower epigastric region, accompanied by vomiting which at times became very distressing. For a few days he could not retain any food by mouth, and it was necessary to give nutrient enemata. He lay doubled up on his side and appeared to have continuous pain, with occasional severe spasms of colic. There was extreme tenderness in the umbilical area and often a sausage-shaped tumour was apparent. The symptoms then abated suddenly and the patient felt quite comfortable, but it took some time for him to recover from the severe prostration. The vomitus was bile-stained, often with a faecal odour, his tongue became dry and coated, and he look very ill. In between the attacks he worked very hard on the farm.

In view of the persistence of the symptoms and after consideration of his earlier history, a radiological examination was carried out on February 3, 1936. This showed that there was some obstructive condition of the duodenum and probably of the jejunum, the actual report being:

(A) Stomach: Normal.

(B) Duodenum: The cap is markedly dilated, the appearance suggesting that there is some block near the junction of the first and second parts.

(C) Intestines: (1) There are loops of dilated small bowel in the duodeno-jejunal area, the appearance suggesting the presence of adhesions. There also appear to be adhesions down towards the terminal portion of the ileum.

(2) There is looping of the distal part of the transverse colon, the appearance suggesting adhesions.

A tracing of one of the films is reproduced, and shows the gross dilatation of the duodenum (see Figure II).

As the attacks were so violent and life was becoming so uncertain, the patient was anxious to try to obtain some relief. Laparotomy was advised in the hope of remedying the obstructions evident in the skiagrams.

The abdomen was opened through a right paramedian supra-umbilical incision. Immediately a grossly distended first part of the duodenum presented. Adhesions between the left lobe of the liver and the old left paramedian scar were divided, and it was then thought that there was an obstructive condition in the upper part of

the second portion of the duodenum. The peritoneum from the gall-bladder passed directly onto the duodenum and had to be divided before the area could be explored. The obstruction then became evident (see Figure III), and appeared to be an old condition, probably a congenital atresia; the site of the lesion was lying well back behind the neck of the gall-bladder, which lay between the dilated first and second parts of the duodenum. On seeking a cause for the dilatation of the second part of the duodenum, another narrowed portion was found at the region of the junction of the second and third parts of the duodenum. This was partly concealed by an adherent and dilated third part of the duodenum, which had to be separated before the anatomical positions could be verified. Again the reason for dilatation of the third part was sought, and on investigating the remainder of the abdominal contents it was found that the caecum and ascending and transverse portions of the colon were quite free except for a thin mesocolon which ran into an

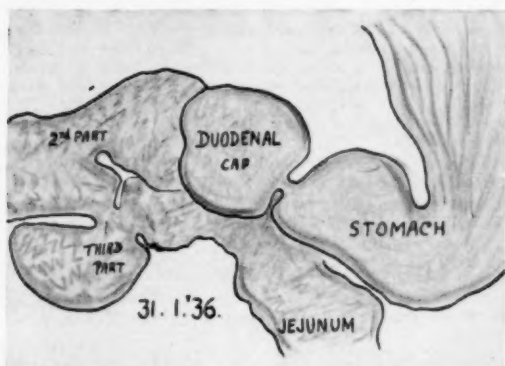


FIGURE II. Tracing of skiagram, January, 1936.

extremely short mesentery of the small intestine, which measured only three centimetres at its base. The caecum was lying with its caput towards the liver, just as is shown in the sketch of the skiagram taken in 1921 (Figure I).

The blood vascular arrangement was exceptional in that the right colic vessels were absent and the ileo-caecal vessels were continuous with the superior mesenteric, the artery being the size of an ordinary lead pencil and the vein about twice this diameter. These ileo-caecal vessels anastomosed with a greatly enlarged middle colic set of blood vessels. All veins were grossly dilated. The entire aggregation of jejunal and ileal coils of the small intestine, with the exception of about forty centimetres of proximal jejunum which was adherent to the posterior abdominal wall, were attached by the small mesenteric base referred to.

Into the elongated and fan-shaped mesentery there was an internal hernia which contained some seven or eight feet of small intestine in a condition of chronic obstruction. The whole of this mass of small intestine was twisted two and a half times around the narrow base of the mesentery, giving an appearance resembling three or four turns of umbilical cord. This torsion was undone and relieved somewhat the vascular congestion in the mesenteric vessels. About a hundred and twenty centimetres from the duodeno-jejunal flexure, the jejunum was fixed by dense, old adhesions to the fan-shaped portion of the mesentery. This resulted in another area of obstruction above which the jejunum and third part of the duodenum were greatly dilated. Upon

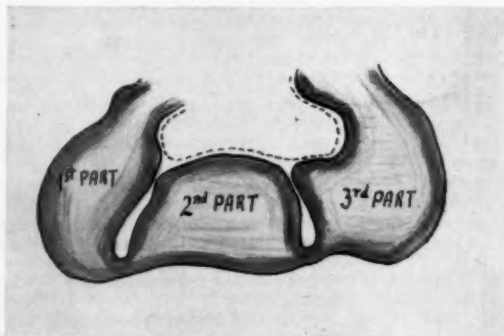


FIGURE III. Sketch of condition found at operation, February, 1936. Note how peritoneal covering obscured recognition of stenosed areas in duodenum.

further examination it was found that the patient had a solitary right kidney, there being no trace of a left kidney or ureter to be palpated. Furthermore, there was no omentum except for a small tag on the left part of the transverse colon.

After the tangle was unravelled and an understanding obtained of the anatomical and pathological peculiarities, the problem was how to remedy them. First the orifice of the hernial sac into the mesentery was closed by suture. Next anastomoses were made between the dilated first and second parts of the duodenum and between the second and third parts. After that an entero-enterostomy was carried out to short-circuit the narrowed area in the jejunum found some hundred and twenty centimetres below the duodeno-jejunal flexure. Next the position of the right flexure of the colon was determined by lifting and rotating the large intestine into position, and then this, the ascending colon and the caecum were fixed in a position in the right flank approximating as near as possible to their normal location. The abdomen was closed after a tube had been inserted through a stab wound in the right flank.

Contrary to expectations, the patient stood the handling and the long operation remarkably well, and twelve hours later expressed himself as very comfortable. He made an uneventful recovery, and stated that at no time after operation had he experienced one-tenth of the discomfort or pain of one of his "attacks".

Since operation he has had a trip to England, and on his return has reported that he has not experienced the slightest discomfort or uneasiness at any time, and has put on 9.4 kilograms (one and half stone) in weight.

Addendum by A. N. Burkitt, Professor of Anatomy, the University of Sydney.

Dr. Poate has suggested that I may like to add some commentary upon the above case.

There are two aspects of this clinical history which are of interest. The first aspect of interest is the combination of congenital abnormalities and the subsequent further disabilities which largely resulted from these abnormalities, and the second, the possibility of some hereditary factor being involved. The congenital abnormalities seem to have been:

(a) Partial persistence of the early embryonic (14-millimetre embryo) stenosis of the duodenum in its first part and probably at the junction of the second and third parts. Dr. Poate is insistent that these stenoses were not due to previous inflammatory adhesions, while owing to their posterior position, they were probably overlooked at the previous operation. The adhesions which resulted in jejunal dilatation were apparently secondary and partly due to inflammation.

(b) Incomplete rotation of the caecum, ascending colon and transverse colon, together with persistence of the corresponding mesocolon and its continuity with a reduced mesentery to the small intestine. Possibly associated with this second abnormality is a third one:

(c) Complete absence of the left kidney and ureter.

The other conditions, such as herniation of the mesentery and rotation of the loops of small intestine round the mesentery were apparently secondary, though probably attributable in part to the congenital conditions. As regards the hereditary aspect of the case, there is some evidence accumulating that similar conditions to these may be due to a lethal or sublethal gene disturbance. However, there is no evidence in this case that any hereditary factor was involved.

Numerous cases of duodenal atresia of varying degree have been recorded; the patients have in some cases died soon after birth, while in some cases the lesion has been associated with other malformations of the alimentary canal, as mentioned by Wakeley.

A considerable volume of literature exists concerning cases of malrotation and abnormalities of fixation of the ascending and transverse colon. I append a list of some important recent papers upon the subject. I am indebted to Dr. G. H. S. Lightoller for some of the facts concerning the earlier history of this case.

Bibliography.

John H. Anderson: "Abnormalities of the Duodenum", *The British Journal of Surgery*, Volume x, 1922, page 316.

Ivar Broman: "*Grundriss der Entwicklungsgeschichte des Menschen*", Munich 1921, page 140.

Norman M. Dott: "Anomalies of Intestinal Rotation: Their Embryology and Surgical Aspects, with a Report of Five Cases", *The British Journal of Surgery*, Volume xi, 1923, page 251.

R. H. Hunter: "A Contribution to the Development of the Duodenum", *Journal of Anatomy*, Volume lxi, 1927, page 206.

Sir A. Keith: "Human Embryology and Morphology", 5th Edition, London, 1933.

Admas A. McConnell and T. Garrett Hardman: "Abnormalities of Fixation of the Ascending Colon: The Relation of Symptoms to Anatomical Findings", *The British Journal of Surgery*, Volume x, 1923, page 532.

Georg Ruge: "Anleitungen zu den Praepieruebungen an der menschlichen Leiche", Leipzig, 1921, page 271.

Cecil P. G. Wakeley: "A Case of Congenital Malformations of the Intestinal Canal", *Journal of Anatomy*, Volume lvii, 1922-1923, page 216.

ACTINOMYCOSIS OF THE VERTEBRAL COLUMN.

By RICHARD FLYNN AND A. D. GILLIES,

Sydney.

ACTINOMYCOSIS of the vertebral column is sufficiently uncommon to warrant the report of an individual case, especially since the one in question presents some unusual features. The history is as follows.

R.C.B., aged thirty-two years, a ship's fireman by trade, was last in perfect health just prior to June 1, 1935, on which date a gangrenous retrocaecal appendix was removed. The peritoneal cavity was drained through an incision well back in the lumbar region. He was five weeks in hospital, the drainage wound healing slowly. A fortnight after his discharge a painful tumour appeared in the right lumbar region, which on incision yielded pus. This incision had never healed properly, and he was admitted to the Royal Prince Alfred Hospital on November 27, 1935, complaining of discharge from, and pain in, his right loin for fourteen weeks; he also complained of pain in the right testis for three days. He had lost 6.3 kilograms (one stone) in weight during this period, and had noticed that he was getting weak; he had an attack of diarrhoea a fortnight before admission, but had had no other symptoms. The pain in his testis he described as dull and aching; he had noticed no swelling. There was nothing relevant in the record of his previous health prior to June 1, 1935.

On examination, the patient was pale and rather wasted, but not cachectic. The scar of the incision in his back made in July, 1935, is well seen in the accompanying illustration (see Figure 1), together with several pouting sinuses from which glairy fluid could be expressed. The margins of the sinuses were dusky red in colour and indurated. The right testis was found to be tender, but not swollen; the epididymis felt normal. A provisional clinical diagnosis of chronic infection due to actinomycosis was made when the patient was first seen by one of us (R.F.) on December 10, 1935.

Investigation.

On December 5, 1935, a skiagram of the spinal column showed no abnormality. Six days later the patient complained of pain in the left side of the chest, and a pleuritic rub was heard in the left axilla. His chest was examined radiographically



FIGURE 1. Photograph of patient's right loin, showing posterior incision and multiple sinuses.

on December 13, and the report was as follows: "Irregular dullness of the right base, possibly pneumonia; the possibility of actinomycosis cannot be excluded. The right lobe of the liver is slightly elevated."

On December 12, 1935, the blood count showed 3,740,000 red cells per cubic millimetre, a haemoglobin value of 62% and a colour index of 0.8, while the leucocytes numbered 20,000 per cubic millimetre, with a neutrophilia of 87% and a shift to the left.

On December 17 the bacteriologist reported on a specimen of pus from one of the sinuses as follows: "A yellow pus containing 'sulphur granules', polymorphonuclear pus cells profuse; Gram-positive filaments resembling *Streptothrix actinomyces* present; Gram-negative bacilli resembling *Bacillus coli communis* present. Sterile on twenty-four-hour incubation on blood agar."

Treatment and Course.

On December 17, 1935, administration of Lugol's iodine solution in milk was commenced, 0.67 cubic centimetre (ten minims) being given thrice daily, which amount was to be increased by 0.4 cubic centimetre (six minims) daily, and the sinuses were syringed daily with *Tinctura Iodi Mitis*. On December 20, 1935, the sinuses were explored without anaesthesia, and were found to extend deeply; accordingly a nitrous oxide and oxygen anaesthetic was administered three days later. On exploration the sinuses were found to be intercommunicating, and one extended downwards in the direction of the caecum. A moderate amount of pus was obtained. The sinuses were laid open and the open wounds packed with vaseline gauze soaked in tincture of iodine. The daily syringing of the wounds with tincture of iodine was continued.

Deep X ray therapy was commenced on January 6, 1936, 1,400 r being given through a filter consisting of two millimetres of copper and one millimetre of aluminium through one port, directly posterior to the right flank, extending from the tenth rib to about five centimetres (two inches) above the iliac crest. The dose was delivered in four exposures of 350 r each.

On February 10, 1936, a further course of deep X ray therapy to the pulmonary lesion was commenced. The treatment was given daily in exposures of 200 or 350 r through four ports, a total amount of 1,400 r being delivered through each. The course was completed on March 3, 1936.

After his admission to hospital the patient developed a cough with mucoid sputum, which was examined on January 13, 1936. The bacteriologist reported: "No pus, streptococci in long chains very profuse. Mouth organisms, including spirochaetes, prominent. Monilia present. Throughout the smear there are scattered short Gram-positive filaments, but I am not prepared to say they are *Streptothrix actinomyces*. No tubercle bacilli detected."

On January 20 the radiologist reported as follows: "The lesion at the right base is much more extensive, and there are now some changes at the left base suggestive of early extension to this lung" (see Figure II). On February 3, 1936, some further slight advance was noted. By February 17, 1936, the lesion showed very definite extension in the right lung, and there was a suspicious area on the left side below the hilum.

On February 20 the patient coughed up what was probably a bronchial cast, which was sent for section to the pathologist, who reported that it had no definite histological structure, whilst the bacteriologist reported: "No pus cells, no evidence of actinomycosis."

A further radiogram was taken on February 27, 1936, and the radiologist reported: "The lesion at both bases appears to have increased slightly since last examination."

On March 12, 1936, the patient first complained of pain in the small of his back. The radiologist reported little change in the lungs, and the wounds on his back appeared to be almost healed by March 30, 1936, while a further radiographic examination on March 31, 1936, showed that the pulmonary lesions were apparently arrested.

A transfusion of 500 cubic centimetres of citrated blood was given on April 7, 1936, and for a period the patient seemed definitely better; but on April 20, 1936, the wound in his back broke down again and discharged pus copiously. His pulmonary lesions were again reported as being stationary on April 27, 1936.

About this time the sinus was probed and bare bone was felt in its depths. On April 23, 1936, the spine was examined radiographically, and the radiologist reported: "There is a compression fracture through the second lumbar vertebra. There is some erosion of the body suggesting that the fracture is a pathological one, consistent with the presence of actinomycosis. There is also some commencing erosion of the tenth and eleventh ribs on the right side." The lateral skiagram is reproduced in Figure III.

The lumbar sinus was injected with lipiodol on May 5, 1936, and the radiologist reported that it ran towards the body of the infected lumbar vertebra.

By this time the patient was receiving 9.2 cubic centimetres (138 minims) of Lugol's iodine solution daily, and his sinus was still being injected with tincture of iodine daily without any signs of iodism developing; but the large oral doses annoyed him, and it was necessary to use constant persuasion to induce him to take them.

By the middle of May his general condition had become poor and he was very lethargic. On May 11, 1936, he was given another transfusion followed by 3.75 cubic centimetres (one fluid drachm) of Lugol's iodine solution in 120 cubic centimetres

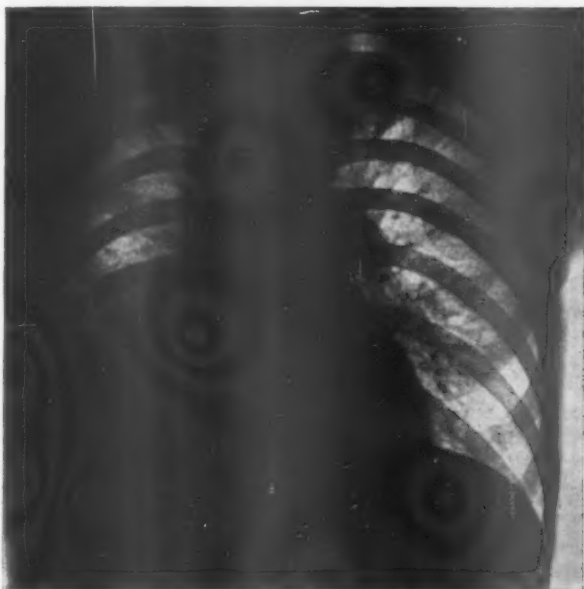


FIGURE II. Radiograph of chest, January 20, 1936.

(four fluid ounces) of normal saline solution intravenously. This injection was repeated on May 18, 1936; on this date *Streptothrix actinomycos* was still present in pus from the sinus. He died on May 23, 1936. During the entire period he was under observation his temperature chart was of the intermittent type.

Ante Mortem Diagnosis.

The patient was never well enough for an exhaustive investigation of the alimentary tract to be carried out, but we were of the opinion, up till about the middle of April, when the bare bone was felt, that he had caecal and pulmonary infection, the former being associated with his attack of appendicitis and the retrocaecal abscess. The spinal lesion was thought to be due to spread by direct continuity from either the pulmonary or the caecal lesion. We also anticipated that the *post mortem* examination would disclose hepatic abscesses.

Post Mortem Examination.

The report of the *post mortem* examination (A.D.G.) was as follows:

"The body is that of a very emaciated young man. There are present sinuses in the right flank and over the spine at about the level of the second lumbar vertebra. A probe passed up both sinuses grates against bare bone.

"There is no excess of free fluid in either pleural cavity. There are some old adhesions present at the apices of both lungs. On the vertebral borders of both lungs there are some very dense adhesions binding the lungs to the vertebral column.

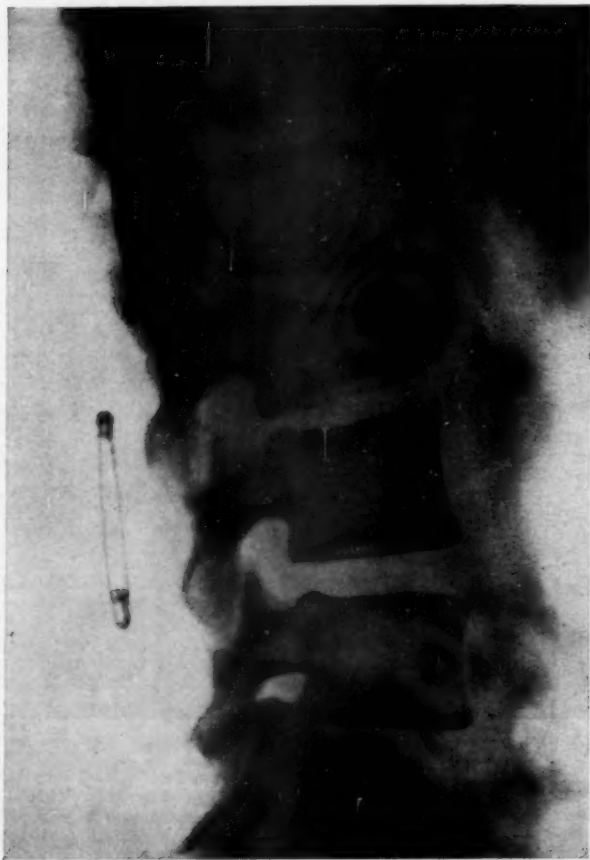


FIGURE III. Radiograph of lumbar spine, lateral view.

On breaking these down, what are apparently sloughing tissue and pus are revealed. The area of these adhesions in each instance is about ten by five centimetres. The left lung is also bound down to the upper aspect of the diaphragm. On section of both lungs near the vertebral borders a gelatinous appearance is disclosed. The honeycomb appearance characteristic of actinomycosis is not visible at the site of section. There is gross enlargement of the pericardium, with an excess of free fluid (about 600 cubic centimetres).

"The visceral pericardium is thickened and white. The cardiac muscle is pale, but otherwise there is no abnormality. The spleen is slightly enlarged and rather firmer than normal in consistency. The liver shows terminal congestion, and on section a hæmangioma about three centimetres in diameter is disclosed. The kidneys show the remains of fetal lobulation. They are very firm in consistency and slightly pale. The œsophagus, stomach and intestines show no abnormality. The caecum, from which the appendix has been removed, is quite free from any adhesions, shows no sign of actinomycosis and looks perfectly normal. The whole alimentary canal has been examined, but no abnormality found. The suprarenal glands are normal. The bladder and genitalia are normal. The brain and spinal cord show no abnormality. There is no involvement of the lymphatic system.

"Examination of the spinal column shows that the bodies of all the vertebræ between the third thoracic and the fourth lumbar are involved in the disease process. They are black in colour, devoid of periosteum, though in places covered by ligaments, rarefied and friable, so that the bone may be easily cut with a knife. The body of the second lumbar vertebra is particularly affected, and collapse has occurred here. The intervertebral cartilages are apparently not affected. Running down behind the right psoas muscle into the thigh is an abscess, and the ilium, where it is in contact with this abscess, is bared of periosteum. The arches and laminae of the vertebræ appear not to be involved in the pathological process."

A sagittal section of the spine was subsequently made with a band saw, and inspection of the cut surface showed numerous small abscesses in the bodies of the vertebræ, from which the *Streptothrix actinomyces* was obtained in large numbers. Figure IV is a photograph of this cut section and shows well the multiple abscesses in the bodies, particularly of the eighth thoracic and the second and fourth lumbar vertebræ. This illustration is of particular interest because it shows well the marked collapse of the second lumbar vertebra and involvement and complete penetration of the intervertebral disk between the first and

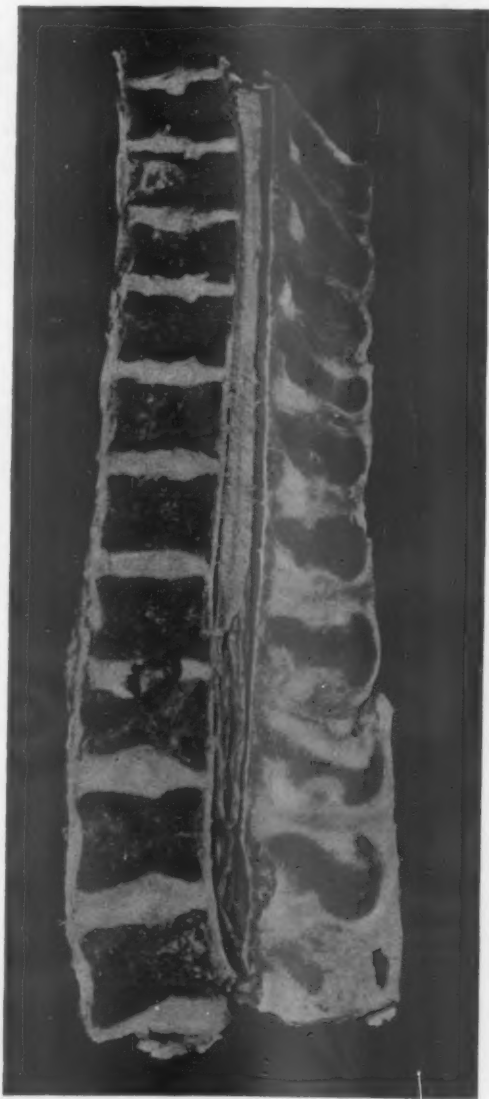


FIGURE IV. Photograph of longitudinal section of spinal column.

second lumbar vertebra. This, however, was not demonstrable in the uncut specimen.

Comment.

The expected involvement of the peritoneum and its contents was not found, and owing to the fact that the most advanced lesion was in the second lumbar vertebra with its associated psoas abscess, we felt that we might have been dealing with a case of primary vertebral actinomycosis. Because of the bilateral involvement of the lungs, where they were adherent to the spine, it seemed that the pulmonary infection was secondary, the right lung being involved before the left.

The difficulty then was that of explaining the source of the spinal infection. The sites of primary infection usually recognized are: (a) the jaws, through carious teeth, (b) the lungs, probably by inhalation, (c) the caecum and (d) the uterus, to which the route of infection is not clear (Gardiner). From these primary lesions spread may occur, as a result of direct continuity or by the blood stream; sometimes even an actinomycotic septicemia develops, with multiple abscesses throughout the body, there being two such cases in the records of the Royal Prince Alfred Hospital.

It is interesting to note that, though the lung condition was of at least five months' duration, it did not show the characteristic racemose appearance of long-standing actinomycosis.

We consider it impossible to be certain of the primary site of infection, but there are several possibilities:

(a) That the primary lesion was in the caecum with appendiceal involvement, that the lung was affected by blood-stream infection, and the spine either by the blood stream or direct continuity. The *post mortem* examination showed no involvement of the caecum, but it may be that the infection here was cured by the intensive therapy. We have evidence that this was effective, in that the progress of the pulmonary lesion was arrested.

(b) That the primary lesion was in the lung, with metastatic spread to the spine. This supposition denies the association of the patient's appendicitis with the actinomycotic infection. The multiplicity of the spinal lesions is in favour, we believe, of metastatic involvement, and the radiological evidence that the tenth and eleventh ribs were also involved is an additional argument in favour of this hypothesis.

(c) That the primary lesion was in the spine with secondary involvement of the lung. The objection to this explanation is the difficulty of understanding how primary spinal infection can occur and its multiplicity of foci. It also ignores the association with the attack of appendicitis.

In favour of the two last-mentioned possibilities is the fact that *post mortem* examination failed to disclose any trace of old inflammation round the caecum.

We are thus uncertain of the site of the primary lesion, but report the case because actinomycosis of the spine is comparatively rare, and because in most articles which have been accessible to us it is stated that the vertebrae do not collapse—a point which is said to differentiate actinomycosis from tuberculous involvement. A further point which we would like to stress is that actinomycosis may invade and even destroy the intervertebral disk; Figure IV demonstrates this point.

Acknowledgement.

We are indebted to Dr. S. S. Gardiner for placing his collection of reprints on spinal actinomycosis at our disposal.

LARGE THORACIC GANGLIO-NEUROMA.

By J. MAXWELL CLARKE,
New Plymouth, N.Z.

THE patient was a girl, aged seventeen years, who was admitted to the New Plymouth Hospital on May 28, 1936. She gave a history of severe pain over the right scapula for one and a half days immediately prior to admission, and of twinges of pain for about two months previously. She was said to have had pleurisy and bronchitis at the age of eleven.

On physical examination the whole of the right lung except the apex was completely dull on percussion, vocal fremitus was absent, and the breath sounds and vocal resonance were very markedly diminished. The clinical diagnosis at this stage



FIGURE I. Skiagram of chest, oblique view. Note the large rounded tumour displacing the trachea and heart.

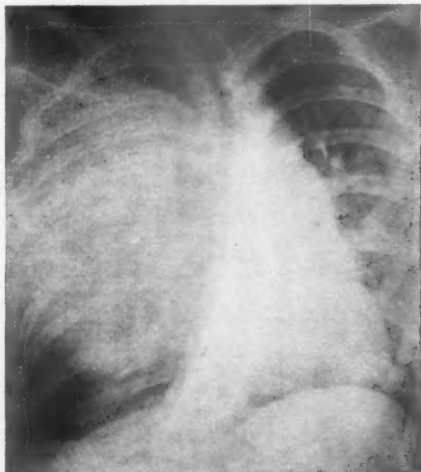


FIGURE II. Skiagram of chest, antero-posterior view. Note the large tumour almost filling the right side of the thoracic cavity, with marked scoliosis and displacement of the trachea and heart.

was one of pleurisy with a large effusion. On needling the chest, under local anaesthesia, no fluid was obtained, and very severe pain was caused after the thorax was entered.

The skiagrams (Figures I and II) indicate fairly clearly the problem which was presented in this case.

Before any operative treatment was carried out, several other tests were done, but without revealing any abnormality, namely, hydatid complement fixation test,



FIGURE III. Photograph of tumour after removal. Weight 1.42 kilograms (three pounds two ounces.)

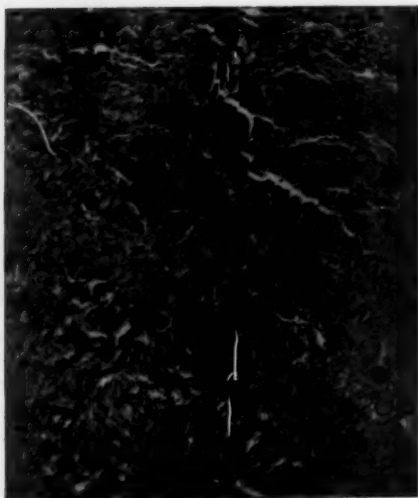


FIGURE IV. Photomicrograph; low power view, showing general features of tumour—isolated ganglion cells, and small groups of ganglion cells lying in tissue which has all the appearance of a neuro-fibroma.



FIGURE V. Photomicrograph; high power view of neuro-fibrils. No tendency to malignancy is apparent.

examination of the sputum, Wassermann test, blood count and blood film examination. Bronchoscopy was performed by Dr. Griffiths, and "Lipiodol" instilled, but this would not enter the right bronchial tree.

Under "Avertin" and nitrous oxide and oxygen anaesthesia the tumour was exposed posterior to the angle of the scapula by resecting a portion of the seventh rib. A small portion of it was removed by the cutting diathermy loop for microscopic examination. The report from Dr. Mercer, of Wellington, indicated a diagnosis of ganglio-neuroma.

Two weeks later, again under "Avertin" and nitrous oxide and oxygen anaesthesia, an attempt was made to remove the tumour. An approach was made through the fifth intercostal space, the ribs immediately above and below the space being divided posteriorly and later resected. An excellent exposure of the middle portion of the tumour was obtained, but unfortunately it was fixed so firmly to the ribs posteriorly and over such a wide area that it was quite impossible to remove it, although it was freed everywhere else. The large veins running on and through the tumour prevented its being transected and thus removed completely. It was decided that the only possible method of dealing with the tumour was to close the incision and attempt its removal through a vertical incision on the posterior aspect of the chest wall, removing wide portions of the seventh, eighth and ninth ribs with the tumour attached. This was duly carried out. The wound was closed, a tube for suction drainage being left through a stab wound. The patient at this stage was suffering severely from shock. A blood transfusion was given in the operating theatre, and she rallied very considerably, but died in the ward within an hour.

At autopsy the tumour was found to have involved the adjacent vertebral bodies, as well as being firmly fixed to the posterior portions of the seventh, eighth and ninth ribs. Its weight was 1.42 kilograms (three pounds two ounces) (Figure III). On naked-eye examination the tumour was lobulated, with large veins running over the surface and through its substance. On section it presented a strong resemblance to a uterine fibromyoma. The microscopic appearances are well shown in Figures IV, V and VI.

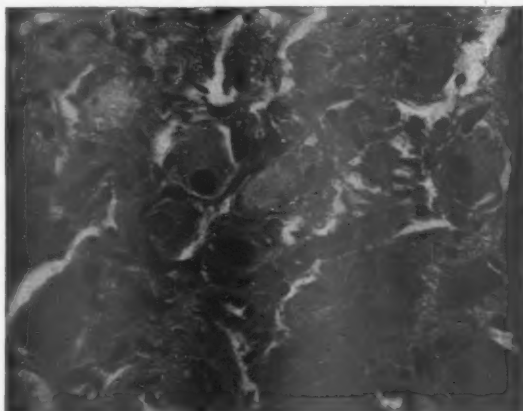


FIGURE VI. Photomicrograph; high power view of ganglion cells which occur all through the tumour.

Discussion.

Ganglio-neuromata are derived from the sympathetic nervous system, arising usually from the thoracic and abdominal sympathetic trunks and ganglia, and from the peripheral perivascular sympathetic plexuses.⁽¹⁾ They may form small, nodular, multiple growths, or large single tumours as in the present case.


There seems to be a big variation in the degree of malignancy exhibited by tumours of this type. Many may be benign, some infiltrate locally, as in the present case, and others definitely have metastases. In most cases they apparently arise from well differentiated nervous tissue, and their characteristics are definitely benign; but on occasions a more primitive type of cell is present. This is an undifferentiated round cell with little cytoplasm. It shows a tendency to rosette formation. It is from this cell that the neurocytoma or neuroblastoma arises, this being a malignant tumour occurring in infants, which at one time was classified as sarcoma. The transitional forms between the innocent and the malignant types indicate a common origin.

The inter-relationship between the histologically malignant and innocent tumours is well illustrated in an interesting case reported by Cushing and Wolbach and quoted by Sophian.⁽²⁾ In a child aged one year they had observed a malignant neuroblastoma in the paravertebral region. Ten years later they removed a recurrence which was then histologically a benign ganglio-neuroma.

References.

⁽¹⁾ J. Ewing: "Neoplastic Diseases", 1928.

⁽²⁾ S. Sophian: "Mediastinal Ganglioneuroma", *Annals of Surgery*, Volume cl, March, 1935, page 827.



The Australian and New Zealand Journal of Surgery.

All articles submitted for publication in this journal must be typewritten and double or treble spacing should be used. Each article should conclude with a brief summary and statement of conclusions. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without any abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given, with full date in each instance.

When illustrations are required, good photographic prints on glossy gaslight paper should be submitted. Line drawings, charts, graphs and so forth should be drawn on thick white paper in indian ink. Authors who are not accustomed to prepare drawings of this kind, are invited to seek the advice of the Editor if they are in any doubt as to the correct procedure. Skiagrams can be reproduced satisfactorily only if good prints or negatives are available.

VOL. VIII.

OCTOBER, 1938.

No. 2.

THE PATHOLOGICALLY-MINDED SURGEON.

THAT surgery is rapidly becoming divided into watertight compartments, each one of which is devoted to some speciality or other, is a matter of common knowledge. Further, surgeons are apt to consider that pathology is outside the realm of their teaching or interest. The question may well be asked: "What is surgery at the present day?" It may be defined as being largely dependent upon a knowledge of anatomy and pathology, and yet, today, there is a tendency to belittle both these important subjects.

Routine pathological examination of every tumour or tissue removed at an operation is imperative and, what is more important, the surgeon himself should examine microscopically the section of the tissue upon which he has operated. This may appear unnecessary, and it may be argued that every appendix which is removed at operation does not require to be examined pathologically. Yet it is only by routine examination of every excised appendix that any real estimation of cases of diverticulitis, carcinoid, actinomycosis and tuberculosis of this organ can be made. Again, the same statement applies to the gall-bladder. Much information has been forthcoming from certain clinics where a complete routine histological examination is made of every excised gall-bladder. Too few examinations, moreover, are made of tissues in the operating theatre itself. To give one example, few

surgeons realize that if the interior of a hydrocele sac of an infant be examined at the time of operation, pyogenic organisms are frequently found. This seems to point to the fact that vaginal hydroceles are in reality infective in origin.

It is perhaps in regard to malignant disease that the surgeon requires to be pathologically-minded. The histological appearance of every tumour he excises should be carefully examined. Too often the surgeon relies on the pathologist's report, with which he is quite content. The surgeon who has seen the clinical manifestations of the growth, and the extent of the lesion at operation, is the only one who can satisfactorily correlate the histological with the clinical findings and add his quota to the final prognosis. The value of the expert whole-time pathologist is not belittled in any way, and his report is of great importance, but it must always be taken in conjunction with the clinical and operative findings.

The pathologically-minded surgeon finds the study of thyreotoxicosis an interesting problem, especially if he contrasts the clinical picture, the estimation of the basal metabolic rate and the histological appearances of the excised gland. If a series of cases are compared it is often seen that the histological findings are at variance with the clinical manifestations. Pictorial records, too, are all-important, and clinical pictures and photomicrographs of every case are today essential. It is an axiom learnt by every medical student that every fracture should be submitted to radiographic examination; and what a wealth of information has been forthcoming from this simple procedure! It is highly probable that similar valuable information would be forthcoming if in every case of thyreotoxicosis the excised gland were submitted to clinical photography and photomicrography. If such a process were adopted, material would be furnished for a further and more complete investigation. We are but playing with the subject at present, and a little more interest on the part of the surgeons would lead to the solution of many problems.

At the present time the majority of surgical clinics have instituted "follow-up" departments which are proving of enormous value. Is it too much to expect that every hospital and clinic should have its own photographic department, where the surgical records would contain not only a clinical photograph, but also a photomicrograph of every tumour excised? It is then, and then only, that these records could be deemed satisfactory. How often valuable clinical material is lost for ever to the profession owing to the incomplete investigation of a case.

CECIL P. G. WAKELEY.

Surgery in Other Countries.

[In this column will be published short résumés of articles likely to be of practical value from Journals published in other countries and not readily accessible to surgeons in Australia and New Zealand.]

KERATOPLASTY.

The Filatow-Marzinkowsky Trephines for Corneal Grafting.

W. P. Filatow: "Die Trepane von Filatow-Marzinkowsky für Hornhautüberpflanzung", *Klinische Monatsblätter für Augenheilkunde*, Volume xcvi, 1936, page 756.

As a preventive measure against wounding of the lens and prolapse of vitreous, the author has for many years used a trephine in conjunction with a guide and guard (see Figures I and II). A through-and-through incision is made through the periphery of the opaque cornea with a Graefe knife or, better, with a special keratome with blunt sides (see Figure III). The guard is inserted through one incision, across the anterior chamber, and out through the other. The guide is screwed onto the plate, the trephine inserted into its guide, and the cutting of the corneal disk carried out without risk of wounding the lens. After the corneal disk is removed the guide is unscrewed from the guard and the latter left in position to prevent prolapse of vitreous until the graft is applied. Should prolapse of vitreous occur when the cornea is being trephined without the assistance of the guide and guard, the author closes the trephine opening with a special obturator, in the form of a disk on a handle, whilst he makes the through-and-through incision and inserts the guard. The obturator is removed, the graft placed in position, the guard withdrawn and the cornea covered with the preformed conjunctival flap.

To obviate the use of the guard, a series of trephines was devised. Ordinarily the anterior chamber is lost as a result of a sudden escape of aqueous. This escapes

round the outside of the cutting edge of the trephine and into the barrel. To close the former

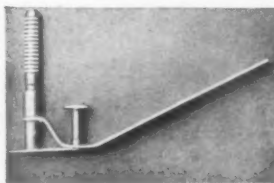


FIGURE I. Trephine and guard assembled.

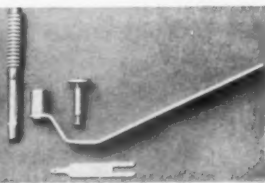


FIGURE II. Trephine and guard, component parts.



FIGURE III. Through-and-through incision with keratome.

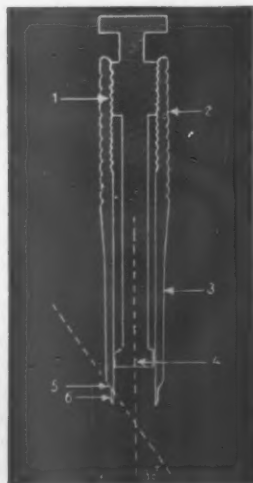


FIGURE IV. Sealed trephine.

channel a ledge is cut round the outside of the instrument at an angle of 30° to the barrel and at a distance of 0.5 millimetre from the cutting edge. Escape of aqueous into the barrel is inhibited by hermetically sealing the latter (see Figures IV and V).

The author does not consider that his improved trephines entirely replace the original form of his instrument. It is to be preferred where vitreous prolapse is likely. When this catastrophe occurs its employment offers the only hope of saving the eye.

In all, the author describes six instruments, the last being a punch-trephine, the guard forming one jaw and the trephine the other (see Figure VI).

The article concludes with a description of a series of six successful cases, in five of which the graft was obtained from *post mortem* material. Two operations were performed with von Hippel's trephine, two with the author's instrument in its original form, and two with modified forms of his rotary trephine.



FIGURE V. Sealed trephine.

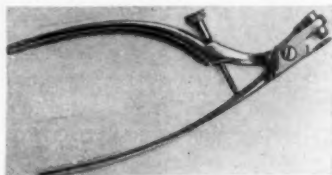


FIGURE VI. "Punch" trephine.

The Use of Post Mortem Material in Keratoplasty.

Zdravko Nizetic: "Leichenhornhaut als Transplantationsmaterial", *Klinische Monatsblätter für Augenheilkunde*, Volume xcvi, 1936, page 756.

In discussing the source of the graft for keratoplasty, the author points out that the possibilities of obtaining living material are necessarily confined. If there is a specific taint present, a malignant neoplasm or sepsis, the cornea must not be used. The surgeon must confine himself to globes enucleated for absolute glaucoma, orbital neoplasm and destructive injury. Murder, suicide, highway and other accidents, poisoning, acute illnesses and post-operative complications provide suitable material, and the autopsy will reveal any contraindication to its use. A search of the literature reveals some experimentation with *post mortem* material, in particular that obtained from children or the new-born. Fuchs preferred the foetal cornea, possibly because of its greater regenerative capacity. Salzer and Elschmig are of the same opinion. At the International Medical Congress in London in 1913, Magitot demonstrated a successful corneal transplant from a foetus of six and a half months. The cornea of the donor was preserved in citrated serum in an ice-chest at a temperature of 5°C . Russian workers, however, were the first to use *post mortem* material as a routine.

The author gives statistics of twenty-four cases in which he used *post mortem* material. In no instance did the graft fail to take. In two cases in which the donor was an eighteen months infant who died as the result of caustic soda poisoning, a streptococcal infection supervened and the grafts became opaque. Of the twenty-four, five remained clear, six were transparent (*durchsichtig*) and thirteen became opaque.

Late Results of Keratoplasty.

S. Ochi: "Ueber zwei Fälle von Dauernd Klar Eingehheilten Hornhautlappen bei der Optischen Keratoplastik", *Klinische Monatsblätter für Augenheilkunde*, Volume xcvi, 1936, page 761.

The first case was that of a woman, aged twenty-nine years, with an opaque cornea following a non-specific kerato-irido-cyclitis. The donor was a live child of three years whose eye had to be removed because of a glioma of the retina. A von Hippel's trephine was used. Four months after the operation the vision was 0.8. Twelve years later the woman wrote to say that vision was as good as ever.

The second case was that of a twenty-two-year-old peasant with a bilateral specific interstitial keratitis. The donor was a child of four years and eight months with a glioma of the retina. Again a von Hippel's trephine of 45 millimetres diameter was used for both donor and host. In neither case were stitches or a conjunctival flap used to hold the graft in place. A year later the corrected vision was 0.3.

KEVIN O'DAY.

SPONDYLOLISTHESIS.

XVIIIe Réunion Annuelle de la Société Française d'Orthopédie, Paris, October 9, 1936, *Revue d'Orthopédie*, Volume xxiii, September, 1936, page 385, and November, 1936, page 626.

P. Mathieu and Demirleau: "Traitement Chirurgical du Spondylolisthésis Dououreux", *Revue d'Orthopédie*, Volume xxiii, July, 1936, page 352.

DURING 1936 several papers appeared in France on spondylolisthesis by P. Mathieu, Froelich, Roederer and others, including a full and important review by Guilleminet, who opened the discussion on this, the principal subject at the annual meeting of the French Orthopædic Society. Many contributions to orthopædic surgery have been made by this important French school in the past, and the present discussion was no exception. Marked cases of the condition under discussion (spondylolisthesis) are uncommon, but the earlier stages (spondylosis) are relatively common. It is therefore a subject of major orthopædic importance, although many orthopædists and surgeons have seen only a few cases. A. Köhler saw none in thirty years, but Meyerding at the Mayo Clinic reported 207 cases from 1918 to 1931, of which 19 were seen between 1918 and 1924 and 188 between 1925 and 1931. The disease will be found to be common enough in any orthopædic department if lateral radiographic examination of the lumbosacral angle is always made in any case of low backache. It will be noted that in Figure I the lateral skiagram shows a normal fifth lumbar vertebra, but in Figure II

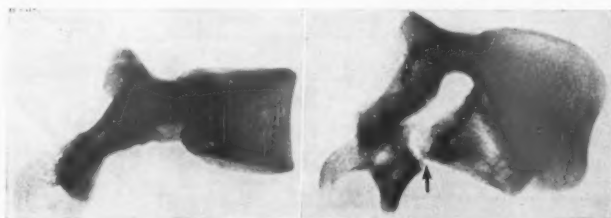


FIGURE I.

FIGURE II.

the important three-quarter oblique picture demonstrates a defect in the pedicle near the root of the articular process. The cause of spondylolisthesis was first suggested by Lambie in 1854, when a fissure was discovered in a fifth lumbar vertebra associated with spondylolisthesis. Thirty years later Neugebauer confirmed this. The fissure may be unilateral, but if it is bilateral the anterior moiety consists of body, pedicles and superior articular processes, and the posterior moiety (posterior arc) of inferior articular processes, laminae and spine. Four *per centum* (Chandler), 2% (Roederer and Glorieux), of skeletons show the abnormality; but Guilleminet found only three in 755 in an anatomy museum, although interesting specimens had been probably removed. This fissuring can involve any lumbar vertebra and even cervical vertebrae. It may be congenital or caused by fracture; the latter lesions are of medico-legal importance and certainly occur. The disease may be latent; even when clinical signs develop, stabilization may develop with natural cure.

Various abnormalities, as for instance enlargements, irregularities and arthritis of the articular processes and joints, were frequently found by Defait in 700 skeletons. *Spina bifida* and fractures of the articular processes occur (Neugebauer, Böhler, and operative proof by Wilson). A unilateral defect causes scoliosis as well as the lordosis caused by the slipping back of the sacrum. The front of the fifth lumbar body is longer than the posterior aspect, leading to a cuneiform body, while the inferior surface of the vertebra becomes excavated by the sacrum, following destruction of the posterior part of the intervertebral disk. The upper portion of the sacrum comes to resemble the *caput femoris*, and a characteristic change after years is a buttress on the front edge of the sacrum as if due to an attempt at spontaneous cure. This latter condition is comparable to the exostosis on the head of the talus in severe flat foot.

Conservative Reduction and Immobilization.—J. Gourdon uses extension in recumbency day and night, with straps around the chest and abdomen and also around the pelvis to overcome lordosis; after ten to fifteen days vertical suspension is employed,

with the use of the two straps to overcome the deformity whilst a plaster case is applied. Numerous successes are reported from the use of the corset, which leads to gradual reduction; but the method is most suitable in children, cases amongst whom are commonly reported in France.

Surgical Treatment.—Anatomical reposition is, except in children, generally impracticable, especially as any such attempts by a dangerous operation on a relatively benign complaint would be unjustified. All posterior lumbo-sacral osteosyntheses have the objection that they do not control the anterior moiety of the fifth lumbar vertebra. The value of certain methods remains to be proved (Burns and P. Mathieu). The first, the simplest, the most commonly employed and also the most to be criticized, is Albee's posterior graft. Its fixation on the sacrum is delicate ("spinous to spinous process"), it acts unmechanically, and it is insufficient as a strong support. It is difficult to fix in firmly on the sacrum. Notwithstanding the good results claimed by some authors, Asbury conceived a bad impression of it, amelioration only occurring in

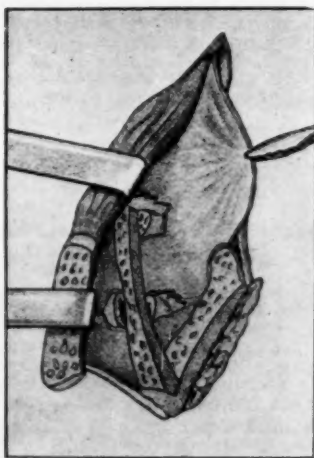


FIGURE III.

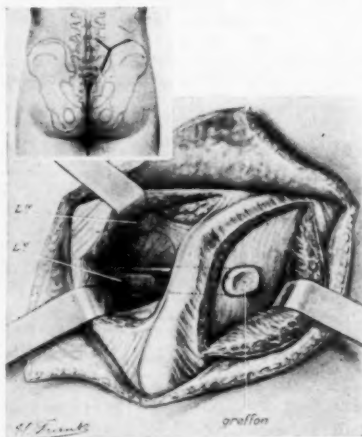


FIGURE IV.

60% to 70% of cases. It is unsuitable for severe cases. Hibb's operation is too severe. The double paraspinous graft on either side of the spinous processes with a broad contact on the surface of the sacrum shows 88% of good results. Arthrodesis of the lumbo-sacral junction is more logical, and was first suggested by Capener and used by B. H. Burns, of London, who transfixed the fifth lumbar vertebral body through the disk into the sacrum with a transvertebral graft from the tibia. The success of Burns's case in a boy, aged fourteen years, is of importance. The operative approach to the lumbo-sacral junction is easy by laparotomy, as is well known in the light of operations on the presacral nerves. Only a number of reported cases is now required to judge its value. Some of the alternative procedures are more dangerous. For example, according to Azema's thesis, the ilio-transverse arthrodesis of Lance and Arousseau by a posterior-median approach is dangerous because the grafts are driven through the ilium into the vertebral bodies, after traversing the pedicles and transverse processes. It appears difficult, impossible in cases of wide separation of the posterior arc, and dangerous to the aortic bifurcation if the graft penetrates too far. Similarly, Mathieu and Demirleau's iliolumbar arthrodesis deserves consideration (see Figure IV). The tibial graft is driven through the ilium into the split transverse processes of the fifth lumbar vertebra, or else the posterior crest of the ilium is used as a graft which fits across the fourth and fifth transverse processes in their posterior aspects, which are broken across so that the graft engages raw bone (see Figure III). Campbell proposed a similar operation. The method is an advantage in the presence of secondary scoliosis. There is a lengthy and important

bibliography after Guilleminet's paper, and following it Froelich (Nancy) spoke in favour of Gourdon's bloodless reduction method, with the use of a special apparatus to exert mechanical corrective pressure.

Glorieux (Bruges) described a special radiographic method of showing early cases impossible to demonstrate in the ordinary lateral radiographic examination, and has eighty cases in his collection. Since this discussion he has published his book in collaboration with Roederer ("*La Spondylolyse et ses Conséquences*", published by Masson, Paris). Roederer (Paris) holds that the true point of treatment exists in two doctrines; one which he calls, rather severely, the American doctrine, is to make a diagnosis and then carry out a radical cure by operative interference, the Albee operation being held in good repute for this course. The speaker naturally does not hold very great account of the verbal statistics of an American surgeon who declared to him that in twenty-six years he had operated upon 1,200 cases of spondylolisthesis! The other, the European doctrine, considers before operation such factors as age, progress of the disease, occupation of the individual, and also the fact that most cases require little treatment. Putti and other Italian orthopædists operate very little. Relatively few cases operated upon have been reported in France, only eighteen in eighty-three described cases by von Schmieden in Germany, and very few in Scandinavia. Pain and continuance of the slipping are indications for operative interference. The most logical procedure is the insertion of a bone peg between the anterior and posterior segments (Capener), *i.e.*, the graft must pass in a postero-anterior direction, but this is illusory because the use of this route is on anatomical grounds contraindicated; also the parts to be nailed are not in a straight line. In regard to pegging the two bodies from in front, also suggested by Capener, it is too early to judge because the only case, that of Burns, was reported seven weeks after the operation. Chalkin (Ekaterinenburg, U.S.S.R.), in his operation, performed ten times for Pott's disease and fractures and six times for spondylolisthesis, generally exposes the vertebral bodies extraperitoneally, resects the lumbo-sacral disk, then places a tibial graft between the fifth lumbar and first sacral vertebral bodies, thereafter nursing the patient in a hammock, which corrects the lumbar lordosis. The operation

has been tried for three years, with no deaths in spondylolisthesis cases. The Japanese, Ito, Tsuchiya and Asomi, also described this procedure for Pott's disease. Roederer himself supports Mathieu's ilio-transverse graft *plus* an Albee graft, although theoretically the former is sufficient. It acts not as a *point d'appui*, but as a calcified iliolumbar ligament preventing further slipping.

The operation most favoured by all speakers was that of Mathieu and Demirleau (see Figure IV); that is, a combination of a supple lumbo-sacral graft with an iliolumbar one on one or both sides. It is simple to perform and not dangerous, according to Mathieu; but all speakers did not agree to this. The iliac crest is rugined of lumbo-sacral muscles on both its inner and outer surfaces. The lumbo-sacral muscles are incised transversely, that is, parallel with the upper limb of the skin incision (careful suturing later is essential to avoid a hæmatoma). This transverse division of muscles caused bleeding and Mathieu avoided it in his later cases. The posterior face of the sacrum is exposed, and also the transverse process of the fifth lumbar vertebra, which, he warns, should be looked for very anterior to the wing of the sacrum, especially in the presence of any marked degree of spondylolisthesis; it can nevertheless always be found. The tibial graft, previously cut with an electric saw, is now driven through a hole in the ilium, which hole should be as far forward as the tip of the transverse process. By means of a fine artist's chisel the transverse process is split, apparently with the chisel thrust along the hole in the ilium. The graft is driven tightly through the ilium into the bivalved transverse process. Recently Zahradnick has supplemented

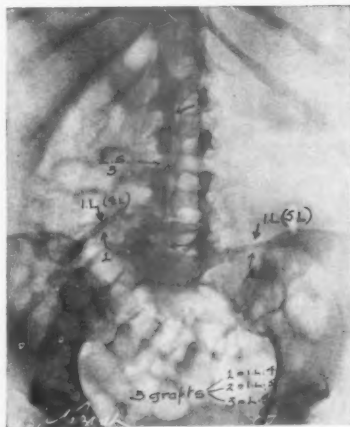


FIGURE V.

I

this procedure by driving the bone peg from the ilium into the body of the vertebra. Mathieu then recommended in addition the lumbo-sacral graft in which one or two tibial strips of bone are rendered pliant, apparently by numerous small transverse saw cuts, and then placed along one or both sides of the transverse processes of the fourth and fifth lumbar vertebrae (which are split and turned over the grafts) and anchored onto a broad surface of the bared sacrum under the muscles, that is, a lumbo-sacral osteosynthesis. Figure V shows both lumbo-sacral and iliolumbar osteosyntheses. Numerous successes of Mathieu's method were reported by the speakers.

THOMAS KING.

CAROTID-CAVERNOUS ANEURYSMS.

Walter E. Dandy: "Carotid-Cavernous Aneurysms (Pulsating Exophthalmos)", *Zentralblatt für Neurochirurgie*, Number 2, 1937, page 77; and *Zentralblatt für Neurochirurgie*, Number 3, 1937, page 165.

In reviewing this article from the ophthalmic standpoint, it is interesting to find frequent references by the author to the works of Benjamin Travers, who was appointed to the staff of Moorfields Eye Hospital in 1810 as its second surgeon, following the death of the founder, John Cunningham Saunders. Dandy further makes reference to the work of Sir William Lawrence, the third surgeon at Moorfields, in relation to the disease under review. In 1809 Travers was the first to treat carotid-cavernous aneurysm by ligation of the common carotid artery, and after one hundred and twenty-eight years this is still the operation of choice, although it gives less than 50% of satisfactory results.

The article, which is divided into nineteen parts, gives a very complete summary of every aspect of the subject and also an extensive bibliography. The author discusses the signs and symptoms of the disease from every aspect, and concludes that exophthalmos, pulsation of the eyeball and a loud murmur are the three cardinal symptoms, and that other disturbances "are diagnostic frills that add little to the fundamental set-up, though they may contribute to the differential diagnosis from lesions that cannot produce them". He next deals with the differential diagnosis from six other conditions, but is of the opinion that "it is difficult to believe that any lesion can offer serious difficulties in differential diagnosis". Among the other ophthalmic signs that he has noticed are chemosis, engorgement of the retinal veins, papilloedema "scarcely indistinguishable from papilloedema of sufferers from intracranial pressure", extraocular palsies in which the abducent nerve is nearly twice as commonly affected as the oculo-motor and trochlear nerves combined, optic atrophy due to pressure upon the optic nerve intracranially by the aneurysm, and, lastly, glaucoma. He also mentions that the trigeminal and facial nerves may become paralysed and so indirectly affect the ophthalmic picture. Pulsating vessels are palpable in the orbit or on the forehead in about one-third of cases. Dandy quotes Sattler's analysis of 219 cases, in which 30% of the sufferers were blind and an additional 20% had less than 10% vision.

He then proceeds to report a series of eight cases from the Johns Hopkins Hospital, Baltimore, between 1905 and the date of publication, and gives a *résumé* of the clinical features and the operative results in this series. In this *résumé* one special paragraph is devoted to the expectancy of vision in untreated cases, and he reaches the conclusion that only about 20% of all cases retain approximately normal vision if they remain untreated. In his own series, owing to immediate operative interference being undertaken, the amount of visual loss in each case was considerably less.

He then fully discusses ligation of the carotid artery for carotid-cavernous aneurysm and comes to the conclusion that 50% of cures in this procedure are all that can be expected, and stresses in more than one instance the absolute necessity of repeated digital occlusion of the common carotid before surgical occlusion is attempted, as any signs of cerebral anaemia from digital occlusion absolutely preclude the surgeon from attempting complete surgical occlusion of the internal carotid. In discussing the effects of ligation he quotes very freely from the work of Benjamin Travers and mentions that Sir William Lawrence was the first to use buried silk sutures in this operation.

One interesting fact mentioned by Dandy in his conclusion is that in one of his cases in which the internal carotid artery was ligated both in the neck and intracranially, the ophthalmic artery still maintained its circulation and no impairment of vision resulted. As a corollary, the aneurysm was not cured, and the external carotid

artery had to be extirpated in the neck; but even this radical procedure left the vision in the homolateral eye unimpaired.

As the major signs of carotid-cavernous aneurysm are mostly confined to the eye, ophthalmic surgeons should find this excellent survey by a surgeon so renowned worthy of their full perusal.

J. BRUCE HAMILTON.

THE BLOOD PHOSPHATASE IN PATIENTS SUFFERING FROM CARCINOMA.

H. Lubenstein: "Über die Phosphatase in Blutplasma bei Menschen mit einer bösartigen Geschwulst", *Zeitschrift für die gesamte experimentelle Medizin*, Volume c, Number 4, April 14, 1937, pages 456 to 467.

H. Baumert: "Hemmung und Aktivierung der Serumphosphatase beim Carcinom", *ibidem*, pages 468 to 476.

Two papers have appeared in the April number of the German journal of experimental medicine which draw attention to the important part played by the phosphatases in the blood plasma of patients who are afflicted with malignant tumours. These papers emanate from the chemical section of the Pathological Institute of the University of Berlin, of which Professor Hinsberg is director.

The first of these papers, by H. Lubenstein, of New York, suggests that the determination of the amount of phosphatase in the plasma may be used to detect the presence of secondary growths in cancer patients, and so to serve as an indication of the advisability or otherwise of removing the primary growth. The following account embodying certain direct quotations from these papers may serve to emphasize the usefulness of information concerning the phosphorus partition and phosphatases in the blood.

Lubenstein points out that the term "phosphatases", as used by him, does not stand for a specific enzyme, but for various phosphatases which possess the common property of being able to detach inorganic phosphate from the various organic phosphoric acid esters. In his introductory paragraph he states: "According to the highly informative researches of Warburg, concerning the peculiar metabolism involved in malignant tumours, workers in the field of neoplasms have now come to recognize that the most important problem is not concerned with the tumour tissue itself, but with the whole organism. In this connexion particular importance attaches to the part played by the enzymes, without the knowledge of which it would be impossible to trace the mechanism of the growth of a malignant tumour. It has been known for some time that the tissue of malignant tumours is particularly rich in enzymes."

Reference is then made to the researches of Köhler and Waldschmidt-Leitz, who have found that the content of phosphatase at the beginning of the growth of the tumour is high, but that it decreases linearly with the age of the tumour. Köhler's experiments appear to show that the phosphatases are particularly concerned with growth processes in the human organism. "In the process of glycolysis, which takes place in the malignant tumour in the absence as well as in the presence of oxygen, and which has been designated by Warburg as the reaction which yields the energy necessary for the growth of the tumour, phosphatase plays an important part. Just as Warburg's maxim for the organisms of higher animals runs, 'There is no growth without glycolysis', so we may also say that there is no growth without an increase of phosphatase. That glycolysis and increased phosphatase activity are two processes that run parallel is borne out by the fact that the two processes are inhibited by the same substances, for example, phlorrhizin and fluorides. An increase of phosphatase is therefore to be expected in rapidly growing tissue or in a rapidly growing organism; this has been confirmed. In the organism of a normal child, a two- to threefold increase has been found in the phosphatase content of the blood plasma as compared with the normal value found in adults (Bodansky and Kay). This manifests itself particularly clearly at the age at which growth is most rapid; the plasma phosphatase is then at its maximum." Animal experiments by Rosahn, Kinard, Kay and Robison confirm these findings.

The phosphatase in the plasma of normal adults has values between 5 and 11 units, as established by Kay and others. Franseen and McLean claim to have found values up to 13 in normal adults. The figures obtained by Lubenstein for normal adults are of the same order. The plasma phosphatase values were obtained in twenty-two cancer patients in whom metastases had been shown to occur. In nineteen cases the values were found to be greater, in some cases much greater, than the value for normal adults;

they ranged from 13.2 to 63.5. In three cases, however, the values were 9.0, 7.6 and 7.3 respectively.

Figures obtained for the phosphatase values in the plasma of carcinomatous patients in whom no metastases had been detected were found to be in the normal range, the actual values lying between 5.6 and 12.9. Results for the phosphatase values in blood plasma taken from the corpses of cancer patients confirmed the deductions made from the values obtained for living patients; that is, high values were present when the primary growth was accompanied by metastases.

The author therefore considers that the determination of the plasma phosphatase may be of great value in diagnosis, since surgical intervention may be recommended only if the phosphatase values do not exceed 13 or at most 15, as then alone is one assured that secondary growths have not yet appeared. When the values exceed 15, the operation may be regarded as offering no prospect of success.

Lubenstein states that: "Nowadays it is generally accepted by workers in the field of tumours that the malignant neoplasm is to be regarded not only as a local disease, but also as a general disease of the body. The increase of phosphatase in the body is therefore not to be left out of consideration in view of the part played by it in many functions of the body." It may be mentioned that phosphatase has recently been proved also to occur in the saliva.

In this connexion a remark in a recent article by J. Needham, of Cambridge, on "Substances Promoting Normal and Abnormal Growth", may be quoted: "It has also been shown that the oxidation products of the active hydrocarbons all inhibit both respiration and glycolysis in every tissue investigated. It is here that we begin to see how work on the chemical stimulating mechanism may link up with work on the metabolic peculiarities of malignant as opposed to ordinary tissue, already pushed so far by the school of Warburg." It is, of course, well known that increased plasma phosphatase values occur also in certain other diseases, such as icterus, rhachitis and *osteitis deformans*. This fact does not, however, detract from the usefulness of the test for differentiating between carcinoma without and carcinoma with metastases in cases where the primary growth is definitely established clinically while metastases cannot yet be detected.

H. L. BROSE.

THE BLOOD PHOSPHATASE IN PREGNANCY.

T. Meranze, D. R. Meranze and M. Rothman: "Blood Phosphatase in Pregnancy", *The American Journal of Obstetrics and Gynecology*, Volume xxxiii, March, 1937, page 443.

THE authors carried out blood phosphatase determinations on 201 cases of normal pregnancy at various stages, including every month of gestation. The average blood phosphatase values were found to be definitely higher than normal in the last two months of pregnancy and during labour. Relatively low phosphatase levels were found in a small number of determinations in cord blood. There was no significant relationship observed between the blood phosphatase levels and the sex of the new-born or the season of the year of their birth. The possibility that the blood phosphatase levels may reflect normal or early abnormal phases of bone metabolism in the mother or fetus is also discussed in the paper.

H. L. BROSE.

SECONDARY HYDATID CYSTS OF THE PLEURA.

Félix Dévé (Rouen): "*L'Echinococcose secondaire de la plèvre*", *Journal de Chirurgie*, Volume xlix, Number 4, April, 1937, pages 497 to 535.

WITH characteristic completeness and exactitude, Dévé, the discoverer of the entity now so widely known as secondary echinococcosis, describes this complication as it affects the pleural cavity and membranes. He limits his study to that of secondary cysts which have arisen from the bursting or leaking of a primary cyst in the vicinity, and of these he could not find records of more than 19, one of which occurred in his own practice, while he saw another when on a visit to the clinic of Professor Costatini of Algiers. Of these 19 cases, 11 were consecutive to a primary lung cyst (3 being post-operative, due to leakage at the time of operation), 5 were consecutive to a liver cyst (2 being post-operative, due to leakage at a transthoracic operation) and 3 to cysts of the spleen, kidney and spine respectively.

In order to estimate the frequency of secondary cyst formation after contamination of the pleura with hydatid material, Dévé collected the records of 104 cases where previous rupture of a primary cyst of the lung had occurred, and found evidence of secondary invasion in 8%. By a lucky chance, the cases of rupture of a liver cyst into the pleural cavity, which he also collected, came to the same total of 104, but in these the proportion showing secondary cysts was only 3%. The liver cyst which bursts into the pleural cavity is usually an old degenerated multivesicular one containing more or less septic bile; severe reactions result, and opportunities for secondary cyst formation are consequently not at all favourable.

By contrast, the liver cyst which bursts into the peritoneal cavity is usually univesicular and clean, and very exceptionally causes severe reactions. In 80% of these abdominal cases secondary cysts are developed. Dévé does not include in this category of intrapleural secondaries cysts which merely bulge from the neighbouring lung, liver, spine *et cetera*.

Of the cases recorded, most of which feature particularly the common complication of pyo-pneumothorax, three are of Australasian origin:

1. Lendon, of Adelaide: "Clinical Lectures on Hydatid Disease of the Lungs", London, 1902, Case VII.
2. Acland: Intercolonial Medical Congress of Australasia, Auckland, February, 1914; incorporated in Barnett's paper read at that congress.
3. Barnett: "Hydatid Pneumothorax", *The British Journal of Surgery*, 1932, Number 76, page 593.

Most of the patients have been middle-aged and of the male sex, and in most cases, strange to say, there has been no definite history of a causative injury and cataclysmic syndrome.

Contamination of the pleural cavity by hydatid fluid, which can so easily occur in the course of an operation or as a result of exploratory puncture, is not necessarily followed by secondary cyst formation. On the contrary, for this to occur the fluid must contain viable scolices or daughter cysts, and these being affected by gravity sink to the bottom of the cyst and so may escape extravasation. If they are extravasated into the pleural cavity, their distribution is also affected by gravity, and the secondary cyst formation is localized accordingly. In three cases, including Acland's, secondary cysts developed in the region of the operation scar as well as within the pleural cavity. The intrapleural cysts usually become embedded in the thickened parietal or visceral pleura, but in some instances they float freely in a serous or purulent pleural effusion. Dévé found that the same conditions could be reproduced experimentally in animals.

The radiographic findings may be very helpful in diagnosis. The spheroidal, clearly outlined shadows are multiple but unilateral, whereas metastatic cysts and multiple primary cysts are bilateral. The shadows are usually parietal in location, as stereoscopic radiography will show, and there may be indications of the site of the originating parent cyst. With a typical hydatid history and clinical signs supplemented by radiographic and laboratory investigations, the correct diagnosis should be established, but it is actually often missed and needle puncture is mistakenly resorted to. If a hydatid cyst in the thorax is revealed by needle puncture, it is wise to draw off as much fluid as will come away and thereby lessen the risk of pleural contamination.

In the treatment of this condition in our present state of knowledge, we have no other means at our disposal than operation, and this may have to be repeated again and again, according to circumstances. Operative measures may include any or all of the following: free incision of the thoracic wall, with rib resection; puncture, incision and evacuation of cysts; "Formolage" in appropriate cases, but not in cysts communicating with a bronchus; dissection of patches of invaded pleura; drainage, which, however, can sometimes be dispensed with; phrenic avulsion and thoracoplasty.

L. E. BARNETT.

OPERATIVE TREATMENT OF HALLUX VALGUS.

K. Lindemann and M. Meyerhoff: "Erfahrungen mit der Teilresektion der Grosszehengrundphalange beim Hallux Valgus", *Zeitschrift für Orthopädie*, Volume lxvii, Part 1, 1937, page 67.

THE results of the two-thirds resection of the first phalanx of the great toe for *hallux valgus* over a period of seven years (1928 to 1934) are evaluated. In the Kiel University Clinic there were eighty operations (thirty-two unilateral and forty-eight bilateral) and sixty-four results (twenty-four unilateral and forty bilateral) were subsequently

examined. The results of the bilateral operations were often subjectively and objectively different, so that they were all grouped together as single cases (one hundred and twenty-eight operations, of which one hundred and four were followed up). Fifty-two results were perfect, that is, the patient was free from any foot trouble. With forty feet the results were functionally fair. The degree of trouble in these could be attributed to the former state of the foot, e.g., splayed-out forefoot *et cetera*. All cases operated upon had slight restriction of movement up to twenty-five degrees, especially lateral and medial movement (passive). Some toes did not look nice on account of exaggerated shortening of the big toe, too much phalanx having been removed, or because they did not lie on the ground on standing. Notwithstanding, the mobility of the toe was functionally sufficient. Only twelve cases showed marked stiffening of the big toe joint, and also in twelve cases there were eight recurrences of the valgus deformity which were classed as bad results. Notwithstanding these twelve objectively bad results, there were only two patients subjectively troubled. The failures were not obvious to the patient because the stiffness in the big toe joint was compensated for by movements in the inter-phalangeal joint, seeing that this joint was brought close to the old metatarso-phalangeal joint by the shortening of the first phalanx. A contraction of the *extensor hallucis longus* tendon was often noted, and was responsible for preventing the toe lying flat on the ground on walking. This could be avoided by lengthening the tendon 1.0 to 1.5 centimetres.

The operation is performed through a medio-dorsal incision. Two-thirds of the first phalanx must be removed and also the exostosis of the metatarsal head, but not, of course, the metatarsal head itself. The big toe is plantar-flexed with a plaster cast; sutures are removed in ten days. The wound became infected in four cases. Arch supports were supplied in most cases, and walking was allowed in three to four weeks. A fifteen-kilometre walk was permitted in two and a half months.

The authors recommend the two-thirds resection in adults as the method of choice. They do not mention *primus metatarsus varus*.

THOMAS KING.

INTRACRANIAL LESIONS IN RETROBULBAR NEURITIS.

Gabriel-Pierre Sourdille: "*Lésions anatomiques endocraniennes dans la névrite rétro-bulbaire*", *Archives d'Ophthalmologie*, January, 1937, page 3.

THE author refers to a patient suffering from bilateral retrobulbar neuritis, who had been subjected to an intranasal operation without benefit by a neurological surgeon who found an arachnoiditis of the chiasmal region. This latter condition came into prominence since its description by Cushing and Balado independently in 1929. As a result of this finding the author attempts to explain the amelioration of symptoms which followed intranasal operative interference in twenty-five of fifty-eight cases of retrobulbar neuritis. The chiasma, intracranial and intracanalicular parts of the optic nerve, are separated from the sphenoidal and posterior ethmoidal sinuses by bony plates, which are often very thin and are traversed by numerous medullary canals connecting the mucous linings of the sinuses with the coats of the nerve and chiasma. The arterial supply of the chiasma, intracranial and intracanalicular parts of the nerve, comes from the anterior cerebral or anterior communicating artery, and participates in any disturbance of the arterial circulation of the chiasmal space. In contradistinction to the intraorbital portion of the nerve, which is supplied by collateral branches from the ophthalmic artery, the posterior portions are supplied from the periphery of the nerve by branches which are end-arteries. Hence the axis of the nerve in these regions, with the centrally situated papillo-macular bundle, is particularly sensitive to any interference with the arterial circulation. This applies especially to the intracanalicular portion, which has a poorer supply than the remainder of the nerve. There is some evidence that the veins are in communication with those of the posterior nasal fossæ by means of small branches through the cribriform plate. Opinions on the existence of direct lymphatic connexions between the posterior nasal fossæ and the arachnoid space are divided.

The author is of the opinion that a mild arachnoiditis of the chiasmal region may follow a mild catarrh of the posterior nasal sinuses. The nasal disease may clear up and leave no trace, but the arachnoiditis persists. Deficient aeration of the nose from malformation may lead to a state of chronic congestion of the chiasmal region, with consequent arachnoiditis. The actual lesion in the coverings of the nerve cannot explain the central scotoma, as one would rather expect a peripheral contraction of the field. The author postulates a secondary arterial spasm, the ill-effects of which

would be felt most in the axial part of the nerve where the papillo-macular bundle lies, and which would manifest itself as a central scotoma. He would explain the beneficial results following intranasal operation as due in the first case to a reflex action of the cervical sympathetic in relaxing the arterial spasm, and in the second, when malformations are remedied, to the decongestive action following improved aeration. From this he concludes that intranasal operation is always indicated in cases of retrobulbar neuritis, as even with syphilis and disseminated sclerosis the nasal disease leads to a *locus minoris resistentiae* in the nerve.

KEVIN O'DAY.

Reviews.

INJURIES AND DISEASES OF THE HIP.

Injuries and Diseases of the Hip. By F. H. ALBEE; 1938. Australia: Angus and Robertson Limited. Super royal 8vo, pp. 298, with 94 figures. Price: 33s. net.

For a long time surgeons have felt the need of a book on the surgery of the hip joint, so it is fitting that this book should be written by Fred H. Albee, of New York. Dr. Albee for over thirty years has been an outstanding orthopaedic surgeon and has done more than anyone to establish the position of the specialist surgeon in bone and joint work. Before the introduction of the electric motor saw, the orthopaedist was considered to be nothing more than a manipulator and brace-maker.

The author states in his preface to the book that an effort has been made to provide a bibliography which is selective and usable rather than complete, and in this he has succeeded.

In the second chapter he describes what he considers a complete armamentarium for the bone surgeon. While most Australian and European surgeons have managed without a traction operating table and an electro-operative bone outfit, the advantages of these instruments are obvious, and those who have employed them would be loath to be without them.

Dr. Albee stresses the importance of familiarity with plaster of Paris and the technique of its application for everyone who undertakes hip surgery. He gives an excellent description of his own operation of bone pegging—an operation which he considers superior to the introduction of the Smith-Petersen nail in fractures of the neck of the femur. Few surgeons possess the necessary tools for shaping a bone graft. This operation does not appear to be as easy to perform accurately as the introduction of the cannulated Smith-Petersen nail with radiographic control. From experience of both procedures it is difficult to believe that a shaped bone graft can secure sufficient immobility of the fragments to ensure union. It would also appear obvious that the results of pressure necrosis on the cancellous osseous tissue of the femoral neck would be greater with a shaped bone graft cut from the cortical bone of the tibia than the three-flanged nail. The success of the bone pegging operation depends on the accurate reduction of the fracture, impaction of the fragments and immobilization of the limb in a double plaster spica as much as, if not more than, on the autogenous bone graft with its biological and physiological factors for promoting callus formation. Albee believes that the flange nail destroys and compresses the cancellous bone within the neck of the femur and is antagonistic to the physiological requirements for union.

This book, as described in the preface, is written with considerable bias towards operative treatment. Hence in the chapter on congenital dislocation of the hip, more space is devoted to the operative than to the manipulative methods of treatment. It is surprising that there is no mention of the Dénucé method of manipulation for this condition. Mention is made, however, of the diagnosis and treatment of dislocation in infancy as described by Putti. Here lies the solution of the problems of the surgery of congenital dislocation of the hip, for if Putti's teaching is followed the problems will not arise.

The operative surgery of arthrodesis for tuberculosis of the hip is, as one would expect, well described. The author describes the methods he employs. Other methods are described in detail, with interesting criticism and observation on each one.

This book of 286 pages is well printed on beautiful paper. The illustrations and reproductions of skiagrams are excellent. It is full of sound advice from a lifetime experience of an active surgeon, and while it has been written chiefly for orthopaedic surgeons, no surgeon who carries out surgery of the hip joint should be without a copy.

APPENDICITIS.

Appendicitis: A Clinical Study. By W. H. BOWEN, M.S., F.R.C.S.; 1937. London: Cambridge University Press. Melbourne: S. Jaboor. Crown 8vo, pp. 213. Price: 7s. 6d. net.

THIS monograph is an interesting survey of the experience of an individual surgeon. The survey of his cases no doubt afforded interest to the author, and those cited will be of value to many readers in that each is a clinical study.

Although some chapters are interesting and instructive, especially those relating to aetiology and severity, and to the obstructed appendix, it is to be regretted that a monograph entitled "Appendicitis: A Clinical Study" should be published in 1937 without any reference to the treatment of paralytic ileus and general peritonitis by gastric drainage by means of a duodenal tube and by continuous intravenous infusion. It is possible that the explanation is that the material seems to have been collected up to 1934. No monograph on this subject in 1937, however, can be authoritative with this omission. The work, moreover, that emphasized the importance of this line of treatment was largely clinical.

The author's record of cases at the Addenbroke Hospital in 1932 is instructive and useful in that he personally saw each appendix. It is very doubtful if other figures, quoted for comparison from previous years, were compiled with similar diligence.

The account of the change-over from medical treatment, morphine and starvation, to early surgical treatment, combined with purgatives and elimination, is another reminder of how often in medicine the pendulum swings too far.

The statement that pronounced fixation of the appendix is uncommon is provocative and dangerous; all surgeons will recall many fixed and difficult extra-caecal appendices, appendices fixed behind the last loop of the ileum, and in stout patients relative fixation from infiltration of a narrow fat mesentery.

It is refreshing to read a chapter that discards pips, pieces of enamel and worms as aetiological factors, and emphasizes infection, whether enterogenous, hæmatogenous or intrinsic. The remarks on the origin of stercoliths and their bearing on the severity of the disease are thoughtful and informative.

In a clinical study more might have been said of the importance of examining centrifugalized specimens of urine and of the value and significance of leucocyte counts.

Many surgeons will rejoice that the author hates the delayed plan of campaign and sponsors the value of drainage, though many will quarrel with his method of "wick drainage". Though there is frequent mention of the use of packs, nothing is said of the use of a suction pump, without which no operation for acute appendicitis should commence.

If the reviewer has differed with several points of view, he appreciates the clinical, indeed even medical, outlook, the care in diagnosis, and the evaluation of the risk of operation. These factors and the study of the patient during convalescence and from the psychological point of view indicate the true clinician.

SURGICAL PATHOLOGY OF DISEASES OF THE NECK.

Surgical Pathology of the Diseases of the Neck. By A. E. HERTZLER, M.D.; 1937. Philadelphia: J. P. Lippincott Company. Super royal 8vo, pp. 237, with 206 illustrations.

THIS book is one of a series of monographs on surgical pathology. The author has done a signal service in collecting together a vast amount of material and presenting it in easily readable form, the racy style and the wealth of illustration making it excellent week-end reading. As he remarks, the book is very largely "a clinical and operating-room study", so that those seeking to improve their knowledge of pathology will probably be disappointed. The experience of the author has been enormous. He has operated upon almost every known lesion in the neck, and his surgical "tips" are worth noting. His remark, that "the present tendency to treat all lesions of the

neck by irradiation without a definite pathologic diagnosis is to be deplored", is one with which we find ourselves in entire agreement. He describes many cases which illustrate this point.

Excellent descriptions of Hodgkin's disease and lymphosarcoma are given. The manifestations of the diseases in the neck are alone considered, however, and there is a complete absence of any attempt to correlate the general constitutional findings. Perhaps such a title as "Morbidity Anatomy of Some Diseases of the Neck" would have been more appropriate.

A long chapter is devoted to lympho-epitheliomata of the neck. This is well worth reading, as it sums up the surgeon's view on that vague group of tumours, usually regarded as secondary to obscure primary lesions in the pyriform fossa and such-like places. This section could have been more rationally placed with that dealing with secondary tumours of the neck. Branchiogenic carcinoma is separately described and not even vaguely correlated with the lympho-epitheliomata. An interesting chapter is devoted to tumours of the carotid body and the neuromata of the neck. The author admits his ignorance of the embryological origin of various cysts in the neck. Such candour is refreshing, but, in a work on pathology, at least some attempt should be made to link up these conditions with aberrations in development. This chapter is not well done. No mention is made of the cholesterol crystals in branchial cysts, or of the relation of the latter to the carotid arteries. Two of the pictures in this section are wrongly labelled, namely, obvious branchial cysts are described as thyroglossal cysts (page 123). Diseases of the salivary glands are described, but mention is not made of fistulae and calculi in this situation. Not all surgeons will agree that most mixed parotid tumours may be completely and permanently removed without damage to the facial nerve. The author confesses that operations on secondary metastases in the neck are futile and he also deprecates the operative treatment of tuberculous glands. A short bibliography is given at the end of each chapter. The illustrations, both of macroscopic and microscopic appearances, are excellent.

The book is a useful aid to the undergraduate, but is hardly complete enough for the post-graduate student working for a higher degree. One cannot escape the conclusion that books like these, while serving a useful purpose, tend to widen the gap between internal medicine and surgery. They should be read by those who have already studied pathology in all its branches, and should not replace, but supplement, the standard text-book.

A SURGEON'S POCKET BOOK.

A Surgeon's Pocket Book. By H. S. SOUTTAR, D.M., M.Ch., F.R.C.S.; Second Edition; 1938. London: W. Heinemann (Medical Books) Limited. Foolscape 8vo, pp. 285. Price: 7s. 6d. net.

If this small book were called "A Surgical Dresser's Pocket Book" it would make a claim which it could well sustain. It is modelled on the author's "Art of Surgery", being a synopsis of the bigger work. There are some excellent plates throughout the book, which in a direct diagrammatic style emphasize the points to be grasped by a student. As a guide for reading and a ready book of reference in the wards, this is a most useful addition to the medical student's library.

Proceedings of the Royal Australasian College of Surgeons.

QUEENSLAND ANNUAL MEETING.

THE annual meeting arranged by the Queensland State Committee was held in Brisbane on August 10 and 11, 1938. The following programme was presented:

Wednesday, August 10, 1938.

At the Physiology Theatre, University of Queensland.

8.15 p.m.—The annual address by H. R. G. Poate, entitled "Anatomy in Surgery".

Thursday, August 11, 1938.

At the Brisbane Hospital.

9 a.m.—Operations by C. M. Lilley and H. S. McLelland.

10.45 a.m.—Presentation of clinical cases by Neville G. Sutton, Alan E. Lee, M. Graham Sutton and J. J. Power.

At the British Medical Association Rooms, Wickham Terrace.

4 p.m.—Five illustrated lectures:

Alan E. Lee: "Certain Aspects of Tumours of the Testicle."

C. E. Wassell: "Congenital and Acquired Deformities of the Nose and Their Correction."

Walter Lockhart Gibson: "Ocular Imbalance."

E. S. Meyers: "The Place of Surgery in the Treatment of Carcinoma of the Tonsil."

R. Graham Brown: "The Importance of Correcting the Nasal Deformity in the Repair of Hare-Lip."

NEW ZEALAND ANNUAL MEETING.

THE annual meeting arranged by the New Zealand Dominion Committee was held in Christchurch on September 1 and 2, 1938. The following programme was presented:

Thursday, September 1, 1938.

At the Christchurch Hospital.

8.30 a.m.—Operations:

Mark Brown: Operation for prolapsus uteri.

J. K. Davidson: Thyroidectomy.

10.15 a.m. to 12.30 p.m.—Demonstration of cases.

2 p.m. to 4.30 p.m.—Water balance in surgery.

1. E. R. Reay: Physiology of water balance.

2. L. A. Bennett: Practical applications in surgery.

3. P. S. Foster: Practical applications in head injuries.

At the Canterbury Club.

6.45 p.m.—Annual dinner.

Friday, September 2, 1938.

At the Christchurch Hospital.

8.30 a.m.—Operations:

E. R. Reay: Prostatectomy.

10.15 a.m. to 12.30 p.m.—Acute osteomyelitis.

1. J. K. Davidson: Treatment of acute osteomyelitis, when to operate and type of operation.
2. W. Bremner: Results.
3. Discussion. Opener, J. L. Will.

2 p.m. to 4.15 p.m.—Ten-minute papers:

1. G. M. F. Barnett: "Injection Treatment of Hæmorrhoids."
 2. Kenneth MacKenzie: "Perineal Hernia."
 3. Sir Louis Barnett: "Hydatid Disease: Some Common Errors in Teaching and Practice."
 4. Walter Fitzgerald: "Some Practical Uses of Kirschner Wire in Traumatic Surgery."
 5. Donald McKenzie: "Ventriculograms."
 6. Roland Fulton: "Parathyroidectomy for Ankylosing Spondylitis (Oppel's Disease)."
 7. J. Hardie Neil: "Relation of Bronchography to Radiology."
 8. H. K. Christie: "Case of Diaphyseal Aclasia with Spinal Complications."
- 4.30 p.m.—Annual Meeting of Fellows.

VICTORIAN ANNUAL MEETING.

THE Victorian State Committee held the annual meeting for the State on September 15 and 16, 1938. The following programme was presented:

Thursday, September 15, 1938.

At the Alfred Hospital.

2.15 p.m.—Operations:

Robert Fowler: Operation for *prolapsed uteri*. Vaginoplasty.

J. M. Buchanan: Operation for *epithelioma vulvæ*: second stage of Bassett's operation.

Clinical demonstration: A case of adreno-genital syndrome: artificial vagina *et cetera*.

E. T. Cato: Bone grafting of tuberculous spine.

C. J. O. Brown: Operation for ureteric calculus.

A. Blaubaum: Operation for multiple stricture of œsophagus.

N. E. H. Box: Bondy's conservative mastoid operation.

C. A. Gardiner and C. W. Nye: Demonstration of ear, nose and throat cases.

J. Ringland Anderson, T. a'B. Travers and W. M. Box: Demonstration of cases of cataract, strabismus, ptosis and trichiasis (K. O'Day also took part, by invitation).

J. Ringland Anderson: A case of retinal glioma treated by radon seeds.

2.15 p.m.—Lecturettes:

H. C. Trumble: "The Application to General Surgery of Lessons Learnt in an Experience of Neurosurgery."

A. J. Trinca: "The Prognostic Value of Microscopic Structures in Carcinoma of the Breast."

L. H. Ball: "The Healing of Appendical Wounds."

Friday, September 16, 1938.

At the Alfred Hospital.

9.30 a.m.—Operations:

H. C. Trumble: Division of sensory root of fifth cranial nerve.

Fay Maclure: Rhinoplasty. Operation for internal derangement of the knee joint.

A. J. Trinca: Radical mastectomy.

2.15 p.m.

Balcombe Quick: Nephrectomy. Operation for Dupuytren's contracture.

L. H. Ball: Anterior gastro-enterostomy (block and gas anaesthesia).

C. H. Hembrow: Operation for inguinal hernia. Demonstration of treatment of chronic backache in gymnasium.

Bryan Foster: Operation for labyrinthitis. Operation for malignant granuloma of nose.

T. G. Wynne: Pachydermia of larynx.

2.15 p.m.—Lectures:

C. A. M. Renou: "Four Cases of *Osteochondritis Dissecans* of the Knee."

C. J. O. Brown: (a) Bronchiectasis: lobectomy. (b) Carcinoma of the lung: pneumonectomy. (c) Secondary pleural echinococcosis. (d) Chronic empyema. (e) Hæmoptysis due to unrecognized foreign body in the lung: lobectomy.

J. Kennedy: "Jejunostomy in the Treatment of Large Gastric Ulcer."

On the evening of Friday, September 16, 1938, Dr. R. A. Willis, M.D., D.Sc., M.R.C.P., delivered the Victorian Annual Lecture at the College. The title of his lecture was: "The Experimental Study of Tissue Transplantation and its Bearings on Surgery."

TASMANIAN STATE ANNUAL MEETING.

THE annual meeting arranged by the Tasmanian State Committee was held in Hobart on October 7 and 8, 1938. The following programme was presented:

Friday, October 7, 1938.

At the Tasmanian Museum.

10.30 a.m.—Annual business meeting of Fellows.

11.15 a.m.—Address: "The Scope of Chest Surgery", by Maurice Susman, Sydney.

At the Royal Hobart Hospital.

2.30 p.m.—Clinical meeting arranged by D. W. L. Parker, of Hobart.

At the Tasmanian Museum.

8.30 p.m.—Symposium on the Surgery of the Head and Neck. Part I. Douglas Miller, Sydney: "Experience with Benign Intracranial Tumours." A. E. Coates, Melbourne: "The Facial Neuralgias."

Saturday, October 8, 1938.

10.30 a.m.—Symposium on the Surgery of the Head and Neck. Part II. A. E. Coates, Melbourne: "Injuries of the Spinal Column." Douglas Miller, Sydney: "The Surgical Sequelæ of Head Injuries."

3 p.m.—Maurice Susman, Sydney: Address—"The Surgical Treatment of Pulmonary Tuberculosis." Illustrated by cinematograph film.

4.30 p.m.—Clifford Craig, Launceston: Address—"A Biological Analysis of Specific Methods of Treatment."

SOUTH AUSTRALIAN ANNUAL MEETING.

THE South Australian State Committee will hold its annual meeting on Wednesday, November 2, 1938. The following programme is to be presented:

At the Anatomy Theatre, University of Adelaide.

8 p.m.—Meeting of Fellows.

8.30 p.m.—Pathological demonstration by Dr. J. Thiersch.

During the week of the annual meeting, Fellows are invited to attend operations performed at the Adelaide and Adelaide Children's Hospitals.

VICTORIAN EVENING MEETINGS.

On the evening of Thursday, August 25, 1938, R. F. O'Sullivan delivered a lecture on "Hysterectomy" at the College. A coloured cinematograph film, illustrating the operation and photographed by Dr. F. Tate, was shown.

On the evening of Friday, October 21, 1938, G. C. Scantlebury delivered a lecture at the College on "Sinusitis".

REGISTRATION OF THE COLLEGE DIPLOMA OF FELLOWSHIP.

THE Council of the College wishes to announce that the Tasmanian Medical Board has granted recognition to the College Diploma of Fellowship as an additionable registrable qualification.

GORDON CRAIG LIBRARY.

IN expressing his appreciation of the library service made available to Fellows from the Gordon Craig Library by the Council, J. Hardie Neil, of Auckland, makes the suggestion that Fellows desirous of presenting books to the Library should forward lists of the books and journals in question to the Secretary, so that he may select any books or journals not at present included in the College collection.

J. Hardie Neil has forwarded a list of books which the widow of the late W. G. Scott, of Auckland, has offered to donate to the College Library.

The Council of the College will appreciate similar action from any other Fellows who may be disposed to assist the College Library.

GORDON CRAIG SCHOLARSHIPS, 1939.

THE Council of the Royal Australasian College of Surgeons invites applications for the Gordon Craig educational and research scholarships for 1939.

Regulations.

The following regulations govern the award of the scholarships:

1. The Council of the Royal Australasian College of Surgeons, having been made the residuary legatee of the estate of the late Gordon Craig, Esq., F.R.A.C.S., one of the founders of the College and a member of its Council, has decided, in accordance with the wishes of the testator, to devote portion of the income of this bequest to the endowment of post-graduate educational and research scholarships, to be known as the Gordon Craig Scholarships.

2. The Council shall award scholarships to such amount in each year as, in its discretion, it thinks fit. Any portion of the income which remains unexpended in any one year may be dealt with as the Council may determine.

3. The Gordon Craig Scholarships for post-graduate education shall be awarded, at the discretion of the Council, to applicants who wish to undergo that course of training which is demanded of candidates for Fellowship of the College. The Gordon Craig Scholarships for surgical research shall be awarded to applicants who, in the opinion of the Council, display special aptitude for this work.

4. Each scholarship shall be awarded for a period of one year, but the Council may, if it thinks proper, renew the award annually for a maximum period of three years.

5. The Council shall have power to determine the manner in which any scholarship shall be paid, and shall have power to cease payment at any time should the work or general conduct of the recipient prove unsatisfactory.

6. The Council shall have complete freedom in determining the value and the number of scholarships. It may, at its discretion, refuse to bestow scholarships in any year. It may also, if it deems fit, award the total amount available to one applicant only. Alternatively, it shall have power to distribute the amount available between any number of applicants in any proportion it considers proper.

7. The Council shall have full power to prescribe conditions in regard to the type of educational or research work to be undertaken and in regard to the place in which this work shall be done.

8. The Council shall have power, at any time, to require any scholar to submit a report upon the work he has done, and shall also have the right to obtain any additional information which may serve to inform it of the manner in which this work has been performed.

9. Applications for scholarships must be lodged, on the prescribed forms, with the Secretary of the College, on or before a date to be advertised from time to time in THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY, *The Medical Journal of Australia*, *The New Zealand Medical Journal*, and to the Deans of the Medical Faculties in Australia and New Zealand.

10. Applicants for Gordon Craig Scholarships, if not eligible for Fellowship of the Royal Australasian College of Surgeons, may be required, at the discretion of the Council, to undertake to proceed to the examination for this diploma upon the completion of the prescribed post-graduate training. Applicants for travelling scholarships must possess a senior surgical qualification recognized by the Council.

11. The Council shall have first claim in regard to the publication of work done by scholars, and may require that such work shall be published in the Journal of the College.

12. Should it so desire, the Council may require post-graduate students or research scholars to repay the amount of the scholarships over a definite period of years without the imposition of any charge for interest.

Forms of Application.

Applications must be made on the prescribed form, which is obtainable from the Secretary of the Royal Australasian College of Surgeons, and must be lodged with him on or before December 31, 1938.

H. G. WHEELER,
Secretary,

Royal Australasian College of Surgeons,
Spring Street, Melbourne, C.I.

Notices.

THE BRITISH POSTGRADUATE MEDICAL SCHOOL.

THE College wishes to draw attention to the announcement of the British Postgraduate Medical School on page xxxii of the advertisements.

NEW DEVELOPMENTS IN SURGICAL EQUIPMENT.

THE attention of Fellows is drawn to pages xx and xxviii among the advertisements, which illustrate some recent developments in surgical equipment. The Editorial Committee is responsible for the selection of the equipment illustrated thereon. The publishers will be pleased, whenever possible, to supply the names and addresses of the manufacturers to anyone requiring such information.

Editorial Notices.

EDITORIAL communications should be addressed to the Chairman of the Editorial Committee, 57 Collins Street, Melbourne, or to any member of the Editorial Committee. It is understood that original articles forwarded for publication are offered to THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY solely, unless the contrary be stated.

Reprints can be supplied at cost price; the minimum number is fifty copies. Orders for reprints must be given when the proof is returned.

Exchange journals should be addressed to the Honorary Librarian, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.I., Victoria, Australia.

Business communications and remittances should be addressed to Butterworth and Co. (Australia) Ltd., 8 O'Connell Street, Sydney.

